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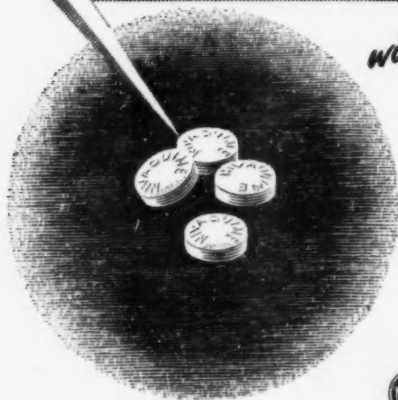


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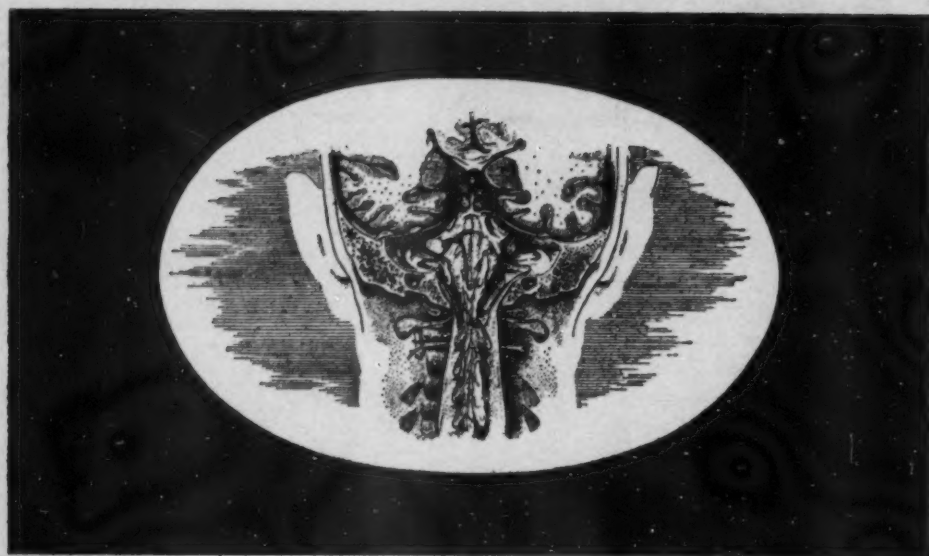
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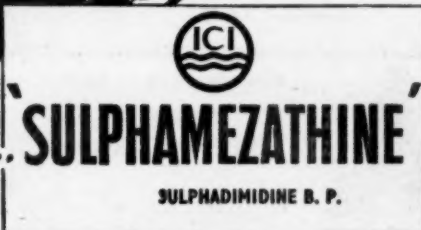
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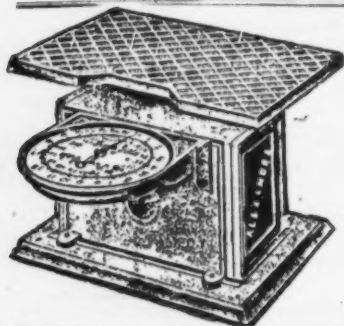
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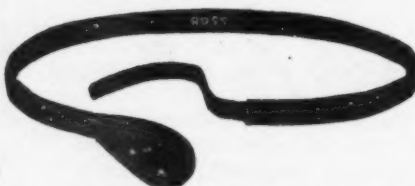
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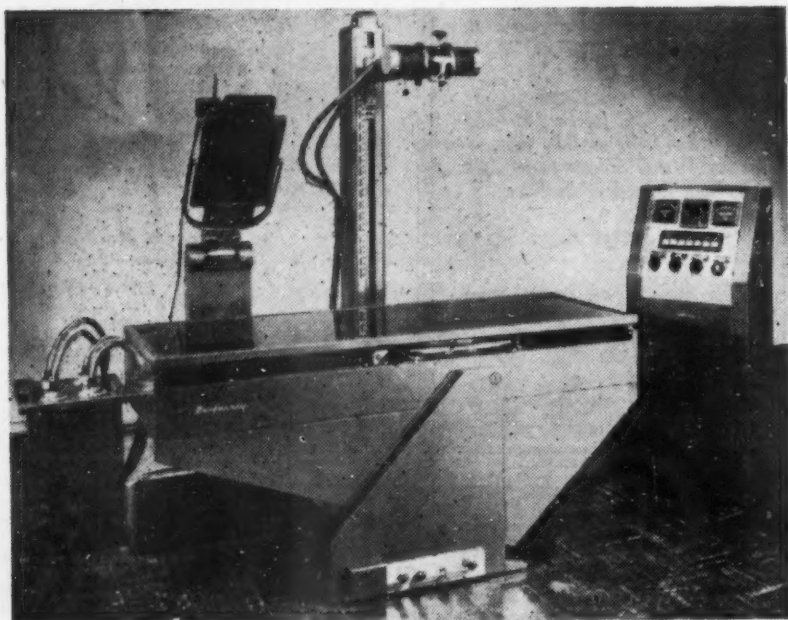
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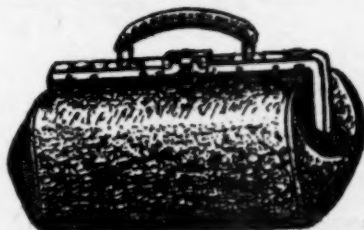
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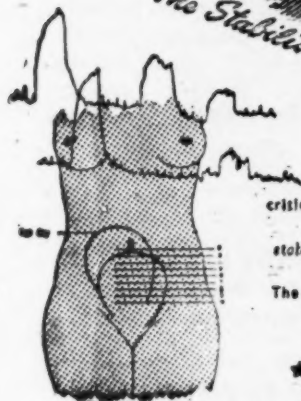
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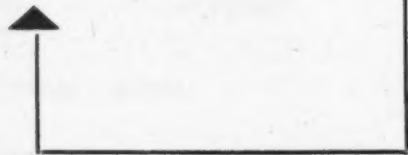
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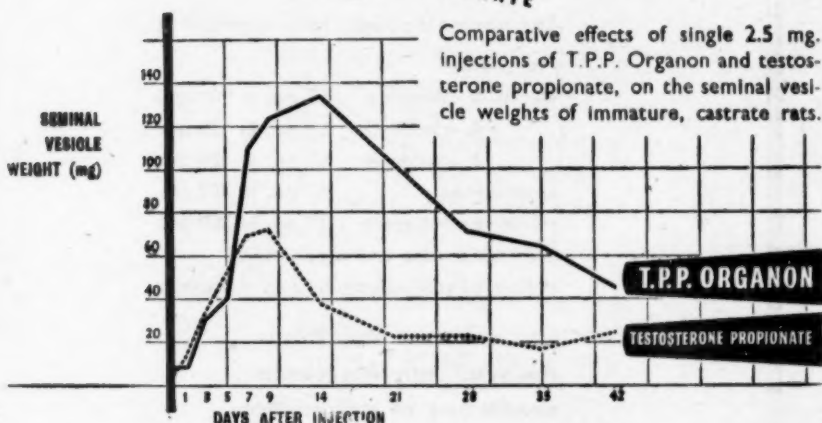
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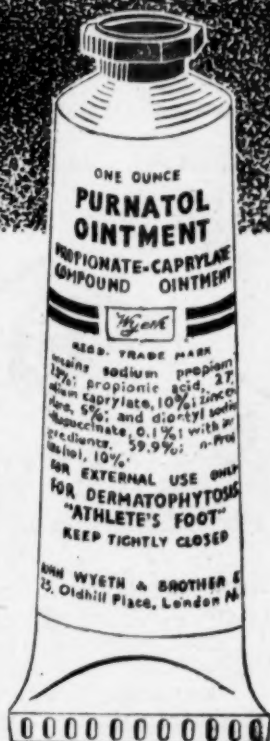
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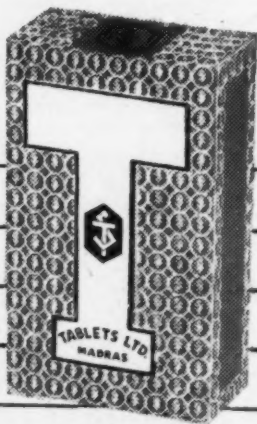
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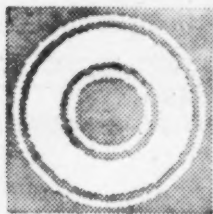


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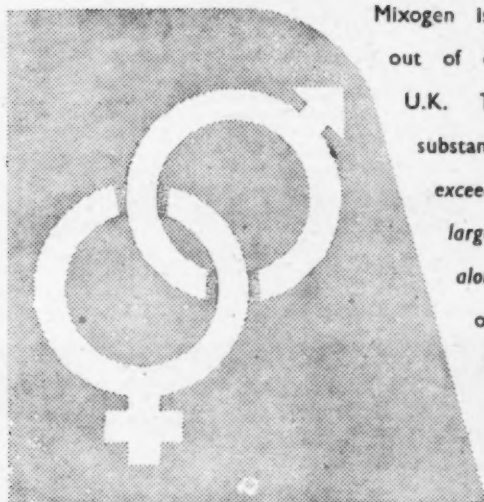


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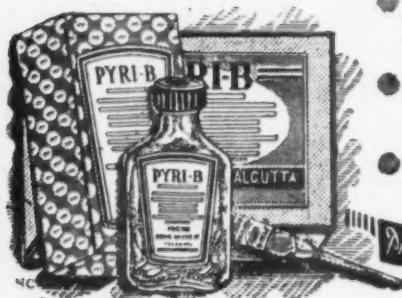
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★ Moore J.E. The Lancet, 1951, 1, 699.

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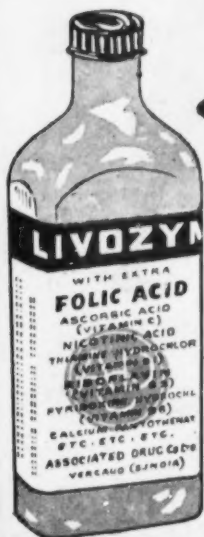
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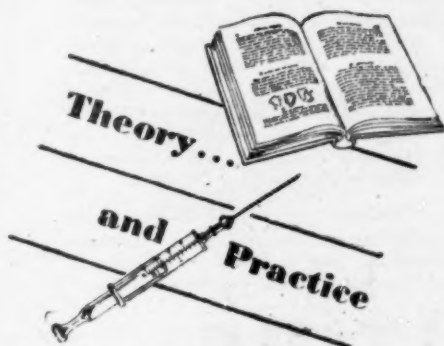


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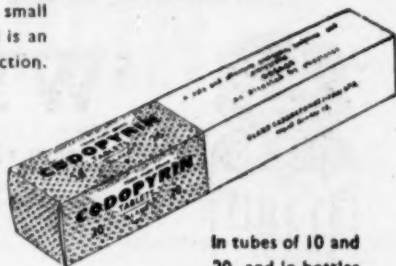
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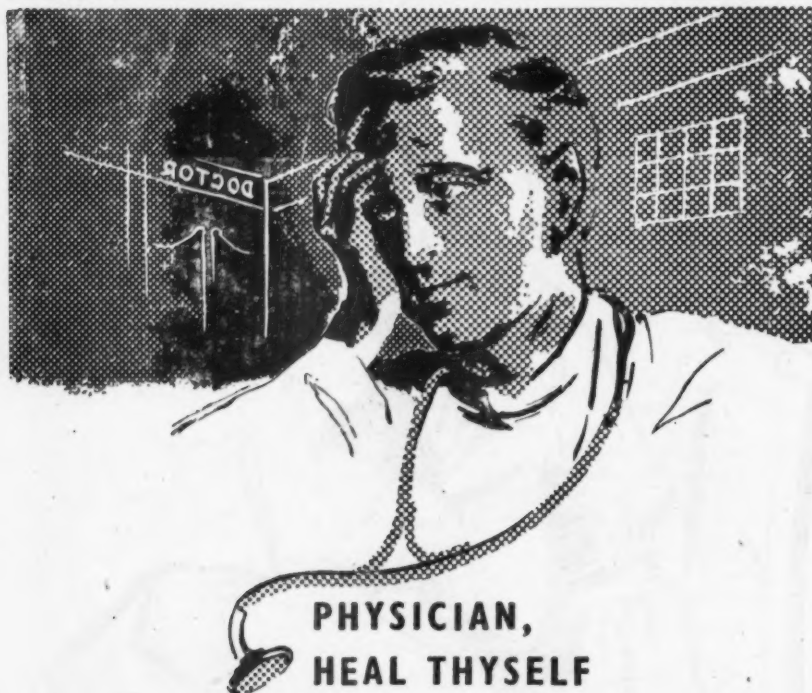
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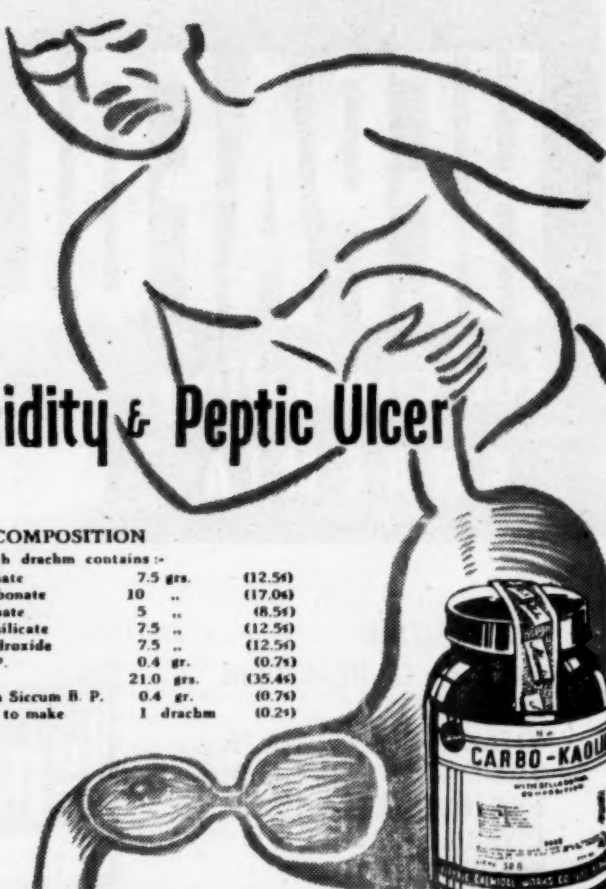
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Original Articles

THE CEREBROSPINAL FLUID*

N. G. TALWALKAR, M.D. (Bom.), M.B.C.P. (Lond.),

Hon. Asst. Physician, Sir J. J. Hospital and Tutor, Grant Medical College, Bombay.

THE cerebrospinal fluid is the fluid which fills the cerebral ventricles and the subarachnoid space.

It is formed by the choroid plexuses in the lateral 3rd and 4th ventricles and reaches the subarachnoid space *via* the foramina of Luschka and Magendie and passes over to the convexities of the brain and the whole extent of subarachnoid space; it is then reabsorbed into the venous system through the arachnoidal villi especially those of the Pachionian bodies in the sagittal sinus.

The precise method of its formation is still not known, but evidence is not wanting to suggest its production by a process of dialysis. Active secretion may also play some part (*e.g.* concentration of certain crystalloids cannot be explained as due to simple filtration).

The total quantity of C.S.F. in a healthy adult varies from 90 to 150 c.c. The rate of its production under natural conditions is unknown; it is influenced by the composition of the blood plasma, the capillary pressure in the choroid plexuses, the permeability of the cells of the plexuses and the pressure of the fluid in the ventricles.

Normal C.S.F. is a clear colourless fluid indistinguishable from water and has a remarkably constant composition. As obtained by lumbar puncture it has the following characteristics:—

* Specially contributed to THE ANTISEPTIC.

(1) Pressure—60 to 150 mms. of C.S.F. (2) Reaction—slightly alkaline. (3) Sp. gr.—1004–07. (4) Appearance—Clear like water. (5) Proteins—20 to 40 mg. % (0.02–0.04 %) mainly albumin. (6) Glucose—50 to 90 mg. %. (7) Chlorides—720 to 750 mg. % (as NaCl). (8) Urea—15–30 mg. %. (9) Cells—0 to 5 per c.mm., mainly endothelial cells and lymphocytes. (10) Organisms—nil. In health, the globulin content is insufficient to give a positive Nonne-apelt or Pandy's test. Langes colloidal gold reaction gives a flat curve and W.R. and Kahn and other tests are negative.

In diseases of central nervous system changes in the physical, chemical, serological and bacteriological features of C.S.F. often give valuable diagnostic and prognostic information. The changes in C.S.F. are pathognomonic in meningitis, syphilitic infection of the brain and spinal cord and subarachnoid hæmorrhage. In spinal block, virus infections of C.N.S. and some intracranial tumours, the examination of C.S.F. may be of great assistance, while many other diseases of C.N.S. such as progressive muscular atrophy, disseminated sclerosis, subacute combined degeneration etc. are not associated with any changes in the C.S.F. at all.

Lumbar puncture is the common method for withdrawing C.S.F. for diagnostic or therapeutic purposes. This is most commonly done through the interspace between the 3rd and 4th or 4th and 5th lumbar spines. In the rare conditions when lumbar puncture is impossible due to local skin condition, disease of vertebræ or adhesion of meninges, C.S.F. may be obtained either by cisternal puncture or by direct tapping of the lateral ventricles through the open anterior fontanelle in infants or through burr holes in the skull in adults.

Pressure of C.S.F. can only be ascertained by actual measurement with a manometer. Estimations based on the rate of flow are often fallacious. A three-way needle with a manometer attachment is useful for this purpose. Before taking readings one must see that the spine is quite horizontal so that the head is neither raised nor lowered, the patient is quite relaxed, the head is not unduly bent forward and the patient is breathing easily and quietly. Normal pressure varies from 60 to 150 mms. of C.S.F. and shows a variation of about 5 to 10 mms. with respiratory movements, falling in inspiration and rising during expiration. Coughing should produce an abrupt rise of about 30 to 50 mms, so also bending of the head or the compression of jugular veins. This is known as Queckenstedt's phenomenon.

In cases of increased intracranial tension the C.S.F. pressure is raised above 150 mms and readings of 300 mms or over may be obtained. In such cases C.S.F. must be withdrawn slowly and in the minimum amount (about 5 to 7 c.c.) necessary for pathological examination; rapid withdrawal of a large quantity may lead to sudden death from formation of medullary pressure cone. Another

useful precaution in these cases is to raise the foot of the bed by 12 to 18 inches so as to reduce the rate of flow of the C.S.F.

Pressure readings at various levels in the cerebrospinal axis will help in the diagnosis of the site of obstruction to the circulation of C.S.F. Thus obstruction of the Foramen of Monro causes a rise in pressure in the corresponding lateral ventricle. In mid-brain lesion, pressure in both the lateral ventricles is raised. In adhesions from meningitis with closure of the Foramina of Luschka and Magendie, the C.S.F. pressure is raised in cisternal as well as lateral ventricles. Obstruction below this point in contrast to the one before produces a rise in C.S.F. pressure beyond the site of obstruction. Thus in spinal obstruction, C.S.F. pressure is raised on lumbar puncture but not in ventricles or in cisterna magna. Puncture of spinal subarachnoid space at higher level than 3rd lumbar vertebra is occasionally done to determine spinal obstruction but carries a great risk of injury to spinal cord.

In spinal block, the C.S.F. pressure is increased below the level of block but the pressure falls rapidly on removal of some C.S.F. and does not again rise rapidly. On coughing or on jugular compression the C.S.F. pressure rises slowly and only a few mms, if the obstruction is incomplete and none at all if the obstruction is complete. These two tests afford valuable evidence of any occlusion of the spinal subarachnoid space.

In case of thrombosis of one of the lateral sinuses, compression of the jugular vein on that side causes no rise in C.S.F. pressure but the pressure on the contralateral jugular vein shows a normal rise in C.S.F. pressure. Thus the alternate compression of the jugular veins is useful in the diagnosis of lateral sinus thrombosis.

Appearance:—Any departure from the normal watery appearance can easily be detected.

(a) Blood-stained fluid may be due to injury to spinal veins during lumbar puncture. In such cases, the first few c.c. of C.S.F. contains more blood than subsequent one, and on standing or centrifuging the red cells settle out, at the bottom leaving the supernatant fluid clear. On the other hand, blood-stained fluid may be due to spontaneous subarachnoid hæmorrhage, cerebral hæmorrhage, which has reached the surface or burst into ventricles, or due to cerebral trauma. In such cases blood is intimately mixed with C.S.F. and is equal in all the specimens. On standing or on centrifuging the supernatant fluid remains pink due to hæmolysis and if a few hours have elapsed since the incident it may show an orange tinge due to the conversion of liberated hæmoglobin into bile pigment by the reticulo-endothelial cells in the meninges.

(b) Xanthochromia or yellow discoloration of C.S.F. is seen in C.S.F. in which blood has escaped a few days previous to the lumbar puncture. This is always seen a few days after an attack of

spontaneous subarachnoid hæmorrhage, and in spinal block when it forms one of the features of Froin's syndrome and is then indicative of rather severe spinal compression. It may sometimes be seen in cases of polyneuritis, cerebral tumour, hæmorrhage, encephalitis and rarely in cerebral arteriosclerosis.

(c) Turbidity of C.S.F. is usually caused by the presence of a large excess of cells and the degree of turbidity increases as the cell count rises, and is thus a characteristic of meningitis. It may vary from a slight opalescence to a frankly purulent fluid. In cases of pneumococcal meningitis the C.S.F. may be so thick that it may not flow out through the needle and may have a greenish tint.

(d) Cobweb, which is a thin pellicle of fibrin clot hanging like a funnel under the free surface is seen only when C.S.F. has been standing for some hours and is seen typically in cases of tuberculous meningitis but is not diagnostic as it may also be seen in poliomyelitis, benign lymphocytic meningitis and in syphilitic meningitis.

Protein content:—Increase in protein content is of great importance and may be seen in many pathological conditions of the central nervous system. It occurs in all cases of meningitis but is particularly marked in case of pyogenic ones. It is, also seen in spinal block and with xanthochromia forms the Froin's syndrome. It is one of the earliest in cases of cerebral abscess. Only a slight increase may be found in cases of poliomyelitis and in most virus diseases of the central nervous system including encephalitis lethargica, in which it may be slight or absent. Isolated and marked rise in C.S.F. protein may be seen in some intracranial tumours when they impinge on the surface of the acoustic neuroma. Also in cases of acute infective polyneuritis (Guillain-Barre syndrome) there is marked rise in C.S.F. protein so much so that it may clot spontaneously.

Increase in proteins without a proportionate increase in cells i.e., albumino cytose dissociation is characteristic of spinal block and cerebral tumour.

Increase in proteins in most cases is manifest in the albumin, but in syphilitic cases the large increase is in globulin content. This is responsible for the colloidal gold reaction in syphilitic cases. Albumin of C.S.F. checks flocculation; excess of globulin overcomes this and produces flocculation in these cases.

Sugar content follows the level of sugar in the blood but with a normal blood sugar, a fall in C.S.F. sugar is an evidence of meningitis of bacterial origin especially the suppurative type in which it may be absent. It gives useful prognostic information—reappearance of sugar being of favourable import. There is no change in sugar in benign lymphocytic choriomeningitis but in encephalitis a rise above normal is often noted. Among the serious meningitis, tuberculous is the one in which the C.S.F. sugar is reduced before reduction in chlorides and so is a good diagnostic aid.

Chloride content is reduced in all types of meningitis especially pyogenic and tuberculous but the reduction is by far most marked in tuberculous meningitis in which it may fall to 600 mgm. or lower. Though formerly considered as diagnostic in tuberculous meningitis it is now known that other conditions may cause fall in chlorides and that fall in sugar is an early and better prognostic guide than the chloride content. C.S.F. chloride is raised when the blood chlorides are raised as in case of renal failure. In benign lymphocytic meningitis C.S.F. chloride remains unaltered.

Urea closely follows the blood level and by itself is not of any importance. It is raised in uræmia.

The cell content is increased in all inflammatory diseases of C.N.S. In pyogenic meningitis there is an enormous increase in the number of cells, the majority being polymorphs. A small number of lymphocytes may also be present and the proportion of these gradually increases as recovery takes place or if the condition becomes chronic. Lymphocytosis is characteristic of tuberculous, benign lymphocytic and syphilitic meningitis. In tuberculous meningitis and also in poliomyelitis in the early stages there may be a high proportion of polymorphs but as the disease progresses the proportion of lymphocytes rises. A mixed pleocytosis is also seen in cases of cerebral and extradural abscess, in sinus thrombosis, and after extensive cerebral softenings. Pleocytosis is also noted in measles even without any neurological symptoms or signs. It may also be the result of intraspinal therapy with serum, penicillin or streptomycin—a point which must be borne in mind when judging prognosis from spinal fluid examination when intraspinal therapy is being given. In syphilitic cases the cell count is a better guide to the prognosis than the protein content because in long-standing infection after treatment, when the infection has been controlled, the cell count reaches normal but the protein remains elevated for a long time.

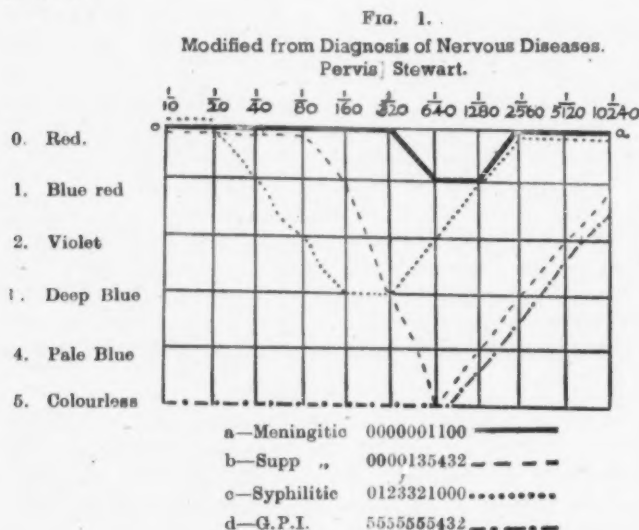
Organisms can usually be easily detected in pyogenic meningitis but in more chronic forms especially in postbasic meningitis in infants meningococci are not usually seen in the lumbar puncture fluid but can be detected only on culture of the ventricular fluid. In tuberculous meningitis it is usually difficult to detect tubercle bacilli in the fluid. Cultural or animal inoculation may be necessary for this purpose. In syphilitic meningitis the virus is not detected in the C.S.F. In torula meningitis the fungus can be seen in C.S.F. but is likely to be missed unless one is aware of it.

Wassermann reaction is positive in all conditions of recent syphilitic disease of the meninges and always in G.P.I. Though often positive in tabes, a negative result is not unusual. The W.R. may however be negative in C.S.F. when it is strongly positive in

blood and this combination is commonly met with meningo-vascular syphilis.

Colloidal Gold and other tests—Langes colloidal gold reaction is a valuable one. The underlying principle that colloidal solutions whether organic or inorganic are protected against precipitation by electrolytes in the presence of a small amount of albumin. The excess of globulin overcomes this and thus produces a precipitate but with albumin beyond a certain point, protection against precipitation sets in.

Normal C.S.F. gives a negative reaction of 0000000000 i.e., no precipitate and no colour change in any of the tubes or 0001110000. In inflammatory conditions precipitation occurs and the colour changes from the original red called as 0 to bluish red, lilac dark purple, pale blue or even to a colourless solution each of these colour changes being represented by numericals 1 to 5. The results are read from left to right beginning with 1/10 dilution. Each pathological fluid has its distinctive 'curve' of precipitation whose maximum lies in a special place in the series of dilutions. (Fig. 1)



In cerebro-spinal syphilis the maximum of the curve occurs in the earlier dilution with a curve 1233210000. This curve is not pathognomonic of syphilis as it may be seen also in disseminated sclerosis.

In G.P.I. a typical gold curve is seen

in 95% of cases with maximum precipitate in the early samples and a sudden drop later 5555554321.

In meningitis a curve is obtained in which its maximum precipitation is shifted very much to the right, for example, 0000001221 and is always to the right of the syphilitic curve. The addition of a little blood also gives a similar curve and therefore, does not obscure a syphilitic curve. These curves, though very useful are

not pathognomonic of a particular disease. However, a normal curve excludes cerebrospinal disease.

TABLE I

	Acute meningitis	Tuberculous meningitis	Benign lymphocytic meningitis	Syphilitic meningitis	Poliomyelitis	Encephalitis	Spinal Block
1. Pressure	++	++	++	++	++ or normal	++	++ Queckenstedt's negative Xanthochromia
2. Appearance	Turbid	Clear or milky Cobweb +	Clear or milky	Usually clear	Usually clear	Clear or pink. No cobweb	
3. Protein	++	++	++	++	At first low. Later rise	Normal	5% or +
4. Glucose	Reduced or absent	Reduced or absent	Normal	Normal	Normal	Increased	Normal
5. Chlorides	Reduced	Much reduced	"	"	"	Normal	"
6. Cells	in 000s especially polys	50 to 500 lymphos	50 to 500 lymphos	50 to 500 lymphos	At first high. Later fall. At first polys, later lymphos	Increased lymphos	"
7. Organisms	Easily detected	Often difficult to show	Not seen	Not seen	Not seen	Nil	Nil
8. Colloidal Gold test	0000135432	0000001100	0000001221	0123321000	0001221000
9. W.R.	Negative	Negative	Negative	Strongly positive	Negative	Negative	Negative

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Magnamycin—The Latest in Antibiotics

Reporting to the recent International Congress on Antibiotics in Buenos Aires, Dr. Hewitt of the California School of Medicine stated that, "probably the greatest significance attaches to the activity of this newest antibiotic, Magnamycin on penicillin—aureomycin—and terramycin—resistant staphylococci." While 27 out of 50 strains of staphylococcus aureus obtained from his patients were resistant to penicillin, only 5 were resistant to Magnamycin. He also showed that extensive therapy with Magnamycin had not produced any significant toxicity or side effects.

Magnamycin has also been used successfully against tonsillitis, pneumonia, amoebic dysentery and local infections such as boils and abscesses. It is not a wide-range antibiotic; it is administered orally and has no cross resistance with any of the common antibiotics. —(M.P.I.B., Feb. 1953).

USE AND DOSAGE OF CHEMOTHERAPEUTIC AND ANTIBIOTIC DRUGS WITH THEIR COMPLICATIONS*

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TWO recent articles in two successive issues of the *British Medical Journal*, one reporting the death of a child following the use of penicillin and the other, an editorial notice of fatalities following the use of chloramphenicol with a warning against the indiscriminate use of the drug, prompted me to record in this communication my observations on the use of these new drugs.

Pharmaceutical science has made great advances in recent years. The century was ushered in by the discovery of a number of chemotherapeutic substances. The salvarsan group started the epoch-making discoveries. Arsenicals have stood the test of time and have given an impetus to further search for synthetic salts. The 2nd and 3rd decades of this century mark the era of biologicals, for conferring active and passive immunity, some of which are still powerful weapons in the fight for disease. From the third decade onwards the now famous chemotherapeutic drugs, e.g., the sulphonamides, sulphones etc. have come into the field. Different combinations of these have been made available to the profession, as the toxicity and limitations of each drug came to be assessed. Then antihistamines, and antitubercular drugs followed in succession. The end of the fourth decade gave us antibiotics like penicillin, streptomycin, chloramphenicol, all of which are of fungal origin. The accidental discovery by Florey of certain cultures of bacteria being destroyed by some unknown substance, was followed up during the war and penicillin was the result. The search for fungal salts has since been in progress and produced some very useful drugs. Further extensive and intensive researches are continually being made in several countries of the world. Of these only chloramphenicol has been synthesised so far. With the therapeutic experience gained by the use of these drugs, limitations to their use have also come to be noted. These limitations were assessed for each drug and unsuitable ones were discarded. The therapeutic action of all these drugs was found to be bacteriostatic except the arsenicals which are bacteriolytic in certain diseases. An attempt is made in this paper to indicate the toxic and other manifestations so far recorded in respect of some of these drugs now in therapeutic use and to sound a note of caution to my brethren in the profession.

The arsenicals.—Salvarsan was the first of the arsenicals to be synthesised. In the early days, to prepare a solution of the drug for use was cumbersome. This difficulty was overcome by the preparation of its sodium salt—Neosalvarsan. Many similar preparations

* Specially contributed to THE ANTISEPTIC.

easy of handling, soon came into the market after World War I. With the increased use of these preparations, it was found that all had their limitations and also produced toxicity of varying degrees on different individuals. The main toxic symptoms noted were:—among others, Herxheimer reaction, dermatitis, kidney damage and encephalitis. Let me give a few instances, which came to my notice. A young boy of 12 was treated with injections of sulpharsenol once in four days, for suspected syphilis; dermatitis of a very severe type developed as evidenced by a thickened eczematous skin, with a foul smelling discharge, and tender and painful ulceration of the mouth. He was in a very depressed mood. The mere suggestion of an injection (sodium thiosulphate for instance), made the boy shudder and walk out of my clinic. The next case was one of pulmonary infarction and encephalitis resulting in death. In this case Neosalvarsan was given thrice, once in four days, in doses of 0.45 gm. to 0.6 gm., and the symptoms appeared within 24 hours after the third injection. Encephalitis occurred in another case when a colleague injected 0.6 gm. Novarsenobillon, on the *fourth* day after the last injection, due to misunderstanding my instructions. Arsenical treatment of filariasis with intravenous Neoarsphenamine was given by a colleague once in four or five days in doses of 0.45 gm. The dermatitis that ensued was so severe that the patient had to be in bed for nearly three months, and had a really very bad time.

Where parenteral arsenical treatment is indicated, I have found the following precautions very helpful:

- (1) The patient is thoroughly examined for the condition of the heart, and liver, for the presence of albumen in the urine and also for his general health. The institution of the specific treatment is deferred in cases of poor general health or damage to any of the organs noted above.

- (2) Whether intravenous or intramuscular, the interval between injections is to be never less than seven days and in exceptional cases six days.

- (3) The urine is examined for albumin before every injection, and the heart, liver and skin for any signs of damage.

- (4) With regard to dosage, not more than 0.45 gm. of Neosalvarsan as individual dose and never more than a total of 4.0 to 4.5 gm. of the drug will be the ideal rule to follow—as in my experience this is the optimum regimen best suited for the average constitution of our countrymen.

The sulphonamides.—Early workers used 1 to 2 gm. initially followed by 1 gm. every four hours, for four to five days or more. This schedule was however, found to be too heavy and so was reduced and with the synthesis of the newer compounds, like sulphamerazine, sulphadiazine, etc., the dosage schedule has been further considerably reduced. American workers have discarded sulphathiazole as being very toxic. I have however, found sulphathiazole

to be very useful in my practice. I usually give 2 gm. a day in divided doses of 0.5 gm., each, the total quantity being 8 to 10 gm. in the course of 4 or 5 days. In exceptional cases I have used 3 gm. on the first day. In children the dosage used has been between 0.75 gm. to 1.5 gm. depending on their age and in infants not more than 0.5 gm. in the course of the day. I have not found it necessary to use the drug for more than four or five days at a stretch. If no relief is noted in the first three days, the drug should be discontinued. The other combinations, sulphadiazine, sulphamerazine need not be used in a dose of more than 1.5 to 2 gm. a day, the smaller dose, being quite adequate in most cases.

The complications following their use vary from a mere urticaria to agranulocytosis, which is the gravest, and therefore, to be prevented if possible. Recovery from this condition is very often doubtful.

The following among others, are the toxic manifestations:—anuria due to blocking of the kidney tubules by crystals of the drug, mental symptoms, anæmia and agranulocytosis. From the results of my experience I suggest the dosage schedule to be followed should be regulated and reduced to suit Indian conditions, for the simple reason that the average Indian's constitution is much below that of the average westerner and cannot stand the high doses administered to the latter.

Penicillin.—This drug which has been hailed as the saviour of mankind has been found to be the least toxic. Yet toxicity has been reported in a few cases and even death due to the administration of the drug has also been recently recorded in America and England. It is by far the most dependable and least toxic of modern medications, and has been extended to a very wide field. I have used it for septic conditions, pneumonia, venereals, certain varieties of dermatitis, early attacks of filaria and others. The use of the procaine salt, has enabled the administration of the drug at longer intervals than the original 4 hour schedule.

The toxic complications noted by me in the course of ten years with the use of this drug, are:—Urticarial rash was seen by me occasionally in cases where the penicillin G powder was used but rarely in procaine penicillin therapy. In some cases this occurred about a week after the cessation of treatment, and so it may not be due to penicillin but may be a reaction due to the underlying septic focus.

In one case of a gangrenous scrotum, the patient developed severe urticaria within two days of penicillin therapy (4 hourly schedule). Therapy was continued and dermatitis soon developed on the extremities, then extending to the entire body. By that time the septic condition had improved and penicillin therapy was stopped. The entire skin from head to foot was desquamated and under antihistamine therapy recovery was effected quickly. It was

subsequently elicited that the patient had a similar dermatitis attack two months previously when he was given penicillin by another doctor, for the commencing inflammation of the scrotum. This experience is a warning to find out the previous history and experience of the patients to this drug.

Streptomycin.—This drug has been in use now for more than seven years. It has been found specific for tuberculosis of all varieties. The dosage in the early days of therapy was very high. With experience, the dosage has been reduced. In my practice, I started with one gm. twice daily for one or two cases and found that though the relief was good, the case did not progress satisfactorily. So I started with one gm. a day divided into two $\frac{1}{2}$ gm. injections or one single injection, usually the latter. This gave better results. Within a year, I reduced the dosage to half a gm. daily, as I found this to be the optimum. Now I am using half a gm. once every alternate day. This has given equally good results, particularly in glandular tuberculosis of both the subdermal and intestinal variety. The patient feels better quickly, the appetite improves and the weight increases. In lung tuberculosis the daily dose of 0.5 gm. is quite effective, and I use the one gm. dose in exceptional cases. So, I enter a strong plea for the use of the smaller dose, and for a total quantity of the drug not exceeding 20 gm. and in exceptional cases 30 gm. in one course.

The toxic manifestations range from urticarial reactions to a complete loss of hearing. With the introduction of dihydrostreptomycin and a lower dosage schedule, reports on the degeneration of the auditory nerve with loss of hearing are getting fewer. I give below the details of an unfortunate experience of mine.

The patient was an elderly lady of 50 years who had lost three children from tuberculosis and herself had bilateral lung disease. With streptomycin and P.A.C. therapy and general tonics, she improved considerably, the total dosage was 42 gm. in the course of three months. Most of the patches cleared up and the last skiagram showed the few small cavities to be healing well. Her weight had increased by 20 lbs., cough, sputum and temperature had completely receded. Her appetite was very good. She had injections for six days in the week. P.A.C. was started with one tablet thrice daily and increased to two tablets twice a day. She could not take more, owing to gastric upset occurring with any increase in the dose. She was perfectly well for a month but complained of noise in the ears from about 10 a.m. to 4 p.m. daily, about a fortnight after the cessation of the therapy. This decreased with the use of B-complex by mouth. She then started getting attacks of dyspnoea and a feeling of sinking, lasting for a few hours, generally at nights. She had about three or four such attacks. When last seen by me she was in a depressed mood but she left my clinic with an assurance that she had improved considerably and was well. Within a week of her last visit, I heard that she had committed suicide by jumping into a well. Perhaps

she developed a psychology that even if she had recovered, her life was not worth living after the loss of her three children; or it may be that due to streptomycin therapy, she developed mental aberrations. I confess I am unable to explain the real cause of the suicide, and so I report this case in order to let the profession know of the existence of a grave possibility like the one recorded here.

The resistance of the tubercle bacillus to this drug has always to be borne in mind. My own view of this resistance is:—When the disease of the lung is characterised by only exudation and consolidation, the result of treatment with this drug is very satisfactory. I suggest that when a cavity exists with fibrous walls and constant accumulation of secretions which are pent up, the tubercle bacilli are not affected by the drug and as such resistance to the drug develops. The possibility of a small percentage of failures with the drug, may therefore, also be found in this explanation which, I submit is well worth serious consideration. For I have always found both clinically and radiologically that a patient without a cavity reacts very well to the drug and the bacilli disappear almost completely, whereas they persist in cases with a cavity. Even with combined P.A.S. or P.A.C. and streptomycin, the possibility of drug-resistance is always to be expected.

P.A.S.—My experience with this drug is limited. I have used a smaller dose than that recommended by other workers, in the few cases in which I tried this drug and nausea and diarrhoea were the toxic reactions noted. With a reduced dose and the simultaneous use of alkalis this can be controlled, if not eliminated.

The toxic reactions noted in a case where combined streptomycin and P.A.S. therapy was given by Jeffrey and Borrie were:—a rise of temperature with rigor, appearance of rash followed by exfoliative dermatitis, enlarged glands, ulceration of the mouth and a high leucocytic count. This was followed by liver and kidney damage. The patient suffered from the toxic reaction for more than a month after which there was recovery. The dosage used was 18 gm. daily of P.A.S. and 1 gm. streptomycin administered for 20 days, when the reactions were noted.

P.A.C.—I have used this drug during the last one year beginning with 1.5 gm. and gradually increasing it to 3 gm. (divided into 3 doses) per day, according to the tolerance of the patient, there was no need to increase the dose in any case. The maximum tolerated dose was given for a period of three to four months, without any toxicity being developed. Usually I give a day's rest once a week or two to 3 days' rest once a fortnight, if the therapy is continuously administered; when combined with streptomycin, I obtained very satisfactory results, the only side-reaction being nausea for a few days at the beginning of treatment, even which can be avoided by suitably adjusting the dose. One other method I adopt is to administer the drug *after food well crushed with milk.*

The sulphones.—This drug synthesised early in the century, fell into disuse, as it was found to be toxic in the somewhat heavy doses in which it was administered. It has now come to be used in leprosy with very good results. I have used this drug in only one case and so far the patient has progressed well. Other workers have also reported that it acts well. The toxic manifestations usually reported have been blood dyscrasias. In suitably adjusted doses, now in use at a very low level and by maintaining the patient's general health in good condition, the drug is able to control the disease.

Chloramphenicol.—The first antibiotic to be synthesised is an aromatic nitro-compound with the formula D(-) threo 2—dichloroacetamido 1-p—nitrophenyl—1, 3—propanediol—(chloramphenicol). The credit for its preparation both in the natural and synthetic states, belongs to the research-workers of Messrs. Parke Davis' Laboratories. It is effective against certain rickettsia and gram-negative bacteria.

The dosage suggested is 50 mg. per kilogram of body weight. The total quantity of drug to be administered varies with the disease. I have used it mostly in enteric cases and in one or two cases of whooping cough. Enteric fever being endemic in our town and district, there was an opportunity for its wide and successful use by nearly all the practitioners; but the cost is prohibitive and beyond the means of most patients. The uniformly good results obtained have, induced even these patients, to use the drug.

I treated nearly a hundred patients. Though the recommended dose was 3 to 4 gm. to be given for 5 to 6 days or longer if necessary, I did not use this high dosage. The average weight of the patients in this place is between 100 lbs. and 130 lbs. with but a very few cases above this level. So, I have never exceeded the dosage schedule of 1.75 gm. on the first day. and 1.5 gm. per day for the next four or five days; and then 1 gm. daily for the subsequent three or four days. The patient starts with 0.5 gm. followed by 0.25 gm. every 4th hour by the clock, day and night, till the temperature is normal for five successive days; remission to normal, usually occurs on the evening of the third day. After five to six days of normal temperature, 1 gm. per day divided into four doses once every six hours, is administered for three days. The total dose administered never exceeds 15 gm. in adults and 6 to 10 gm. in children; the interval between successive doses is longer in children, being usually once in six hours after the first one or two days. This lower dose alone has produced uniformly good results in all my 100 cases. Only two cases relapsed in my series and these were controlled with a second course. The following is the usual course that the disease takes once the administration of the drug is begun. The temperature begins to defervesce within 24 hours and comes down to normal in 72 hours and remains so, until

convalescence is established. The usual precautions relating to diet, hydrotherapy and bed-rest are all observed rigorously. With the early diagnosis and institution of the specific treatment with the drug, there were hardly any complications, not even mild bronchitis. Haemorrhage did not occur in any case, though the literature mentions that this complication cannot be avoided. When the drug has been stopped, urotropin is administered for a week, and there have been no relapses. When pneumonia complicates the disease, penicillin has to be administered, as chloromycetin is usually unable to deal with the complication.

The reactions noted were very few. Marked sweating starts from the 2nd day of touching normal and continues for a week or ten days; general weakness bordering on prostration was seen in a few cases; one case showed severe mental symptoms. The patient was a hefty strong and well-built medical man; he touched normal as usual on the 3rd day and continued normal for six days. In the early hours of the morning on the seventh day he started delirium and soon became violent, and it was with the greatest difficulty that his relations could control him. When I saw him on that day, he was very violent, and uncontrollable and had hallucinations. Injection of $\frac{1}{4}$ grain of morphia, did him no good; So, a further $\frac{1}{4}$ grain was given which controlled him. When he woke up after six hours the violence and delirium had left him and subsequent recovery was uneventful. A colleague consulted me about a girl of 12 years with similar symptoms and the child was controlled within eight hours by a suitable dose of Pot. bromide given by mouth.

I treated two cases of whooping cough one of which was the three months' old child of the above patient. The child had suffered for nearly two weeks without the disease being controlled. The child was given $\frac{1}{6}$ of the 0.25 gm. capsule three times a day, and with the very first dose the child had slept well with abatement of symptoms. With only two capsules, on the whole, the child got complete relief. In the other case about four capsules were required to produce complete relief. No complications were noted in either case. I consider that the rapid relief was due to the smaller doses used. The fatal cases reported in the foreign medical press relate to children who received fairly heavy doses, administered for some length of time; and the complication reported was agranulocytosis. The nitrophenyl radical in the drug is believed to be responsible for the agranulocytosis and subsequent fatality. That the phenyl group gives rise to blood dyscrasias is well known. With the nitrophenyl group as one of the radicals in the drug, there is every likelihood of such changes in the blood. Early workers with this drug have reported marked prostration and anæmia, which however, improved with the discontinuance of the drug. Obviously therefore, the drug should be used in smaller doses and for the optimum period necessary to effect a cure.

I have had no personal experience of the other newer antibiotics like Terramycin, Aureomycin and I.N.H. But from the reports I have seen in the medical press, considerable care and circumspection would appear to be necessary for the proper selection and administration of these drugs.

Accordingly I venture to make the following observations for the benefit of my brother practitioners of medicine :—

1. Whenever a new drug is placed on the market, the reports of original work published from time to time in medical journals that are available to the members through their respective medical associations should first be carefully studied. The composition, action, therapeutics, dosage and toxic reactions should be thoroughly understood before the new drug is tried by them.

2. The practitioner should always keep an open mind about its reactions on his patients and carefully watch, study and record them, as also untoward reactions if any, which should be promptly dealt with.

3. Short accounts of the toxic reactions met with and how he managed to counter them should be communicated to the medical journals for publication so as to benefit his brethren in the profession.

4. The patient and his relations should be warned not to expect quick relief or magical cures nor feel disappointed when the results are not satisfactory.

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1. British Medical Journal, p. 70, 12th July, 1952. P. 136, 19th July, 1952.
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Treatment of Ascariasis in Children with Hetrazan Syrup

Høekenga of the Medical Department of T. R. Co. and U. F. Co. of La Lima, Honduras, utilized a highly palatable preparation of Hetrazan Syrup (Lederle Laboratories Division of the American Cyanamid Co. supplied the syrup) for the treatment of ascariasis in children. He was prompted to do so, as a result of a communication (Loughlin *et al*, *Lancet*, 261: pp. 1197-1200, 1951) indicating that elsewhere in the Caribbean area, administration of such a syrup reduced the egg count by more than 90 per cent. The dose used was 6 mg. of diethyl-carbamazine (Hetrazan) per lb of body weight, once a day for 4 days.

125 children of Honduras, were treated for ascariasis with Hetrazan in a syrup which contained 30 mg. of the drug in each c.c. Of the dosages employed the most effective was 12 mg. per pound of body weight daily for 4 days; in 80 per cent of 30 cases this amount resulted in the absence of ascaris eggs from concentrated (zinc sulphate flotation method) stools on both of two post-treatment examinations. Smaller amounts gave less satisfactory results. 18 mg. per pound of body weight appeared to be too toxic.—(*Am. Jour. Trop. Med.*, 1:4, July 1952).

DRUG THERAPY IN CORONARY DISEASE*

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Introduction.—The use of drugs in coronary disease resolves itself into the treatment of several symptoms and functional disorders of the heart and circulation. There are at present no chemical agents that act directly and deal with the structural abnormality in the heart muscle and its blood vessels; drugs have obviously no place in the treatment of coronary disease if the subject is free of symptoms or of manifest disorders of function. The one exception is cardiovascular syphilis with involvement of the coronary vessels.

The specific objectives to which therapeutics is directed in coronary disease are: (1) pain; (2) nervous symptoms, apprehension, anxiety and restlessness; (3) congestive heart failure; (4) paroxysmal dyspnoea; (5) shock; (6) disorders of heart rhythm (auricular fibrillation or flutter and ventricular tachycardia); and (7) embolism.

The following drugs or other programmes will find their respective places in tackling the problems arising in coronary diseases: (a) opium alkaloids; (b) oxygen therapy; (c) sedatives; (d) xanthines; (e) digitalis; (f) quinidine; (g) diuretics; (h) nitrites; (i) papaverine; (j) iodides; (k) dicoumarol; (l) emergency measures and convalescence; and (m) rehabilitation.

Opium alkaloids.—Morphine is the drug of choice in the treatment of classic coronary thrombosis, the condition first described by Herrick, in which there is excruciating and agonising pain, mental anguish and terror of impending death. Morphine relieves the pain by raising the threshold at the centre and not by dilatation of the coronary vessels—as a matter of fact morphine tends to constrict the coronary bed by vagal stimulation. The beneficial effects of morphine in coronary disease are that (1) it relieves pain; (2) abolishes the tendency to move about; and (3) gives rise to euphoria characteristic of this narcotic.

The sharp edge of pain which is intractable with a single dose of morphine, may be dulled with repeated doses—but not by heroic doses such as depress respiration. 1/4 gr. morphine may be given subcutaneously and repeated at half-hour intervals till the pain is abolished or reduced to a minimum. Sometimes intravenous administration may be desirable. The interval between the doses should not be shorter than half-an-hour and more than 1 gr. in twelve hours is not desirable.

Morphine sometimes complicates the course of coronary thrombosis. It promotes constipation with abdominal distension

* Read at a monthly meeting of the Malabar Branch of the Indian Medical Association.

and urinary retention through spasm of the bladder sphincter. It also causes vomiting, often for several hours. The violent muscular effort is a source of danger in coronary thrombosis, besides creating confusion in deciding whether the vomiting is due to the drug or to the coronary thrombosis itself. Morphine causes powerful vagal stimulation which, renders the heart more susceptible to ventricular ectopic rhythms.

In the mild forms of coronary thrombosis, codeine in doses of $\frac{1}{2}$ to 1 gr. may be used with advantage. Also, it is used instead of morphine when the latter has been used for two or three days. Dilaudid may be used in doses of $\frac{1}{20}$ gr. Habit formation should be guarded against. Pantopon is used by some but has no particular advantage. In cases of intolerance to morphia, as evidenced by vomiting or '*Cat reaction*', a combination in capsule form of $\frac{1}{2}$ to 1 gr. codeine with $\frac{1}{150}$ gr. of scopolamine hydrobromide will sometimes provide a satisfactory substitute.

Oxygen therapy.—In cases where there is pain that is resistant to other measures, placing the patient in a tent with O_2 of about 50% concentration is sometimes helpful, particularly in cases with cyanosis.

Sedatives.—Sedatives play an important role in the management of patients with coronary disease. The popular ones are Barbitol, Phenobarbital, Amytal or Pentobarbital sodium. By reducing nervous excitability, barbiturates are effective in reducing the number and severity of the attacks of coronary insufficiency. The fear and anxiety which intensify the distress of acute coronary thrombosis may be controlled by barbiturates, as also the restlessness which involves a dangerously large expenditure of physical energy in this condition. The sedatives may reduce the susceptibility of the heart to ectopic rhythms and thereby help to prevent ventricular tachycardia, which is a complication in coronary thrombosis.

It is unwise to exceed $\frac{1}{4}$ to $\frac{1}{2}$ gr. Phenobarbital t.i.d. or from $\frac{3}{4}$ to $1\frac{1}{2}$ gr. of Pentobarbital sodium or analogous doses of similar drugs. Larger doses cause stupor associated with motor unrest defeating the very purpose for which they are administered.

Xanthines.—The purine bases, Theobromine and Theophylline are extensively employed in the treatment of coronary artery disease. They are used either alone or mixed with ethylenediamine or sodium acetate or sodium salicylate. The most popular one is Aminophylline.

It was in 1895 that Askanazy first used the xanthines. Within these sixty years, their use has gained such great momentum that it is improbable now to see a cardiac patient with complaint of 'pain in the chest' to have escaped a course of treatment with

aminophylline some time or other. In 1902, Breuer heralded it as 'the most praiseworthy achievement of the decade'.

Dr. Harry Gold, the noted pharmacologist and cardiologist is of opinion that patients with coronary disease and effort-angina obtain no more relief of their cardiac pain from these compounds than they do from sugar of milk administered in the same way. He concludes that aminophylline, theocalcine, theobromine with sodium salicylate or any of the other xanthine derivatives exert no action that is useful in the routine treatment of cardiac pain or myocardial infarction. On the contrary, they are extremely useful as diuretics in congestive cardiac failure. Respiratory depression with Cheyne-Stokes respiration occurring in the course of renal or cardiac failure often shows dramatic improvement from the intravenous injection of from 0.25 to 0.5 gr. of aminophylline.

Digitalis.—The value of digitalis in coronary artery disease is a much debated point. Should it be given as a routine? Does it ever do any good? Is it uniformly dangerous or are there some cases in which it is especially suitable, or particularly dangerous? The two general indications for digitalis therapy are:—(1) cardiac failure; and (2) certain disorders of rhythm with or without cardiac failure namely, auricular fibrillation or auricular flutter and possibly paroxysmal tachycardia.

Primary failure of the heart muscle manifests itself either as right ventricular failure giving rise to dyspnoea, orthopnoea, distended veins of the neck, enlarged liver, pulmonary congestion, oedema and ascites or as left ventricular failure giving rise to recurring paroxysms of dyspnoea or pulmonary oedema. Frequently the clinical picture represents a combination of the two. If a patient has auricular fibrillation with a rapid heart-rate or cardiac failure with early or late coronary thrombosis as the cause or as an accompanying condition, the patient should be digitalised. Not all patients with coronary thrombosis should be digitalised.

Some dangerous effects have been ascribed to the use of digitalis:—

(a) "Digitalis, by increasing the force of the heart's contraction, will promote a tendency to rupture the tenuous tissue of the infarct." Digitalis does not raise the intraventricular pressure. How can it promote rupture of the ventricle without increasing the pressure by which it is ruptured? The danger, therefore, lies not in giving digitalis but in withholding it from a patient who has heart failure in the course of coronary thrombosis.

(b) "Digitalis increases the work of the heart".—In the absence of failure, digitalis tends to decrease cardiac output and diminish the work of the heart; but, in failure the case is one of increased output and increased work. The increased work after digitalis is the result of an improved capacity for work rather than

an increased demand for work. The heart becomes more efficient as a result of the digitalis administration.

(c) "Digitalis constricts the coronary vessels and further diminishes the coronary blood flow."—Diminution of coronary flow is indicated by an increase in the frequency or severity of flow cardiac pain—But in no patient treated, was the course of the pain influenced by even toxic doses of digitalis.

(d) "Digitalis might predispose the 'coronary thrombosed' heart to ventricular tachycardia".—In fact, it requires 50% to 75% of the fatal dose of digitalis to precipitate ventricular tachycardia. The problem is the desirability of giving smaller doses to treat cardiac failure in patients with coronary thrombosis.

A satisfactory dosage schedule in the average case of auricular fibrillation or heart failure in coronary thrombosis is about 6 grs. (4 cat-units) of digitalis leaf daily for 3 days followed by 3 to 4½ grs. a day as long as necessary. The patient should be watched carefully for the appearance of or an increase in the number of ventricular premature beats, which is an indication for reducing the dose.

Quinidine.—Quinidine controls troublesome premature contractions and abolishes auricular flutter, auricular fibrillation and ventricular tachycardia. Ventricular tachycardia is the disorder most feared, after coronary thrombosis, especially of the most severe grades with 250 beats per minute. Levine says quinidine might be useful in small doses as a prophylactic in all cases of coronary thrombosis; but Gold does not subscribe to this view. To patients who present premature ventricular contractions after a coronary thrombosis, an oral dose of 5 grs. of quinidine sulphate may be given three times daily and the dose may be increased if necessary by 5 grs. daily until the abnormal beats disappear or minor toxic symptoms appear. The intravenous injection of quinidine is dangerous. Quinidine should be used only if it appears necessary to expedite the restoration of the normal rhythm. If signs of congestive failure are present, digitalis is to be preferred for slowing the ventricular rate in auricular fibrillation and to restore the normal rhythm in auricular flutter.

Diuretics.—The indications for diuretics overlap those for digitalis. They are useful for the control of congestive heart failure and attacks of paroxysmal dyspnea. An intravenous injection of 1 c.c. of salyrgan or mercupurine two or three times a week with or without a daily dose of 5 or 6 gm. of ammonium nitrate if and when necessary, often proves very helpful.

Nitrites.—Nitrites are the drugs of choice in effort-angina, as they dilate all peripheral arterioles including the coronary vessels, and produce, a physiologic form of relief by abolishing the mechanism that causes pain. The disagreeable effects of nitrites are the flush, headache, a sensation of throbbing and tension in the head,

palpitation and in excessive doses, collapse. In angina of effort, anticipatory treatment is the best. As many as 50 tablets or more of glyceryl trinitrate may be given, without risk. The frequent use of nitrites will not result in habit formation or reduced efficacy. When nitrites fail to relieve a particular pain, in a person who had previously obtained relief from their use, the particular attack would prove to be due to coronary occlusion and should therefore, be confirmed by an EKG. In acute coronary thrombosis, nitrites afford relief only occasionally. The pain appears to arise from the completely infarcted area and from the adjacent muscle with impaired circulation. Nitrites will afford relief in the latter. There is a hazard in the use of nitrites in the acute phase of coronary thrombosis; the nitrites reflexly (from fall of blood-pressure) stimulate the cardiac accelerators, which may precipitate dangerous ectopic tachycardias; and by further lowering the already lowered blood pressure, may impair the blood-flow to the rest of the coronary bed, since the efficiency of the coronary circulation depends on an adequate level of systemic pressure.

Papaverine.—Papaverine in $\frac{1}{2}$ to 1 gr. doses exerts a mild general depressant action and appears to have little value in coronary disease.

Iodides.—While iodides are extensively used in the treatment of coronary sclerosis, they are useless in coronary disease, except in the presence of syphilis.

Dicoumarol and Heparin.—Over a third of the patients with myocardial infarction have thrombo-embolic phenomena occurring in the first six weeks after the infarction. Recently a special Council of the American Heart Association studied the use of anticoagulants in patients with infarction and recommended the routine use of anticoagulants in all patients who have suffered an infarction. The anticoagulants used are Heparin and Dicoumarol in 300 mg. doses. The patient should be hospitalised and prothrombin time accurately estimated.

Emergency measures and convalescence.—Shock, pulmonary oedema, Adams-Stokes' attacks and acute collapse are certain emergency situations that occur in coronary thrombosis. Epinephrine is occasionally helpful in acute pulmonary oedema when the systemic pressure is low, and in Adams-Stokes' attacks. The danger of ventricular tachycardia has however, to be kept in mind. Posterior pituitary extracts should not be used at all since they are coronary vasopressors. Caffein-sodium benzoate, metrazol or coramine are of value in acute collapse. During the attack complete physical and mental rest is essential. Frequent tiring examinations are undesirable. The use of enemas should be avoided for one or more days even in the absence of bowel movements, and an enema on the third or fourth day will be better tolerated. Dehydration should

be combated with 1000 c.c. of normal saline. Some prefer 100 c.c. of 50% glucose solution. Some give 250 c.c. of plasma intravenously.

Good nursing is very important. The diet in the early days should preferably be liquids. A low calorie diet (500–800 calories a day) is advocated by Dr. Master. The patient should be kept at rest for four to eight weeks. He may be permitted to use the commode instead of the bedpan. Dock and Harrison advocate treatment of the patient with coronary thrombosis, out of bed and in a chair. Ambulatory patients should be allowed to return gradually to their normal activities. Prolonged bed-rest is often unnecessary, as it may lead to the development of complications like hypostatic pneumonia, prostatic obstruction, renal-stone-formation and phlebitis with pulmonary embolism.

The patient during convalescence should be advised to:—
(a) do things slowly and 'take it easy'; (b) obtain a sedentary job free from excitement; (c) have rest at recumbency for one hour after lunch and after dinner; (d) avoid tobacco in any form; (e) take light diet divided into 4 or 5 meals; (f) have 9 to 10 hours' bed rest at nights; (g) take very limited quantities of alcohol, if used to it; (h) reduce obesity; and (i) avoid a chill atmosphere, especially after meals.

Rehabilitation of the coronary cardiac.—That coronary disease means either an early death in harness or complete invalidism is a needlessly pessimistic attitude. 'Thousands of patients have succumbed in spirit in the fear of death stalking them—they dare not raise a finger in work or active play again but sit at home or in their cars or at the movies or on the sands of the seashore watching the hours and days go past. They forget that Nature is doing her best and very often also successfully to re-establish by collaterals or by re-canalisation, or by both, an adequate coronary circulation.

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Clinical Studies of Pentaquine

Layman treated 23 veterans who had contracted vivax malaria in the Pacific area with 60 mg. pentaquine base and 2 g. quinine daily, for 14 days; no case of proven recurrence occurred among the patients treated by this regimen.

He is of the opinion that the pentaquine-quinine routine of therapy is curative in a high percentage of cases and that under carefully supervised conditions, the toxicity of this therapy does not contraindicate its use.—(*Journal Kentucky State Med. Assoc.*, July 1952, pp. 305/7).

DYSPNŒA IN CHILDREN*

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DYSPNŒA in children is one of the symptoms in many diseases which need a thorough investigation for arriving at a quick diagnosis. Loss of time may mean loss of life. The investigations required being simple in nature, need not cause delay in arriving at a diagnosis.

The more common causes of dyspnœa in the order of their occurrence are:—(a) primary and secondary bronchopneumonia; (b) bronchitis; (c) asthma; (d) diphtheria; (e) whooping cough. The less common causes may be: (1) lobar pneumonia; (2) congestive cardiac failure due to congenital cardiac diseases, mitral disease etc.; (3) the anæmias; (4) tuberculosis of the lungs; (5) pleural effusion and empyema; (6) retropharyngeal abscess; (7) foreign bodies in the respiratory passages. Acute œdema of the larynx, mediastinal glands, ascitis etc. may rarely cause dyspnœa.

When a case presents itself with acute dyspnœa, a rapid systematic examination and a good look into the nose and throat are absolutely necessary. Often a single examination fails to point the cause of the malady and it is then necessary to examine the child again and again until the cause has been detected. When the dyspnœa is continuous, accompanied by pyrexia, rapid breathing and cyanosis, bronchopneumonia and lobar pneumonia must first be thought of. To differentiate between the two is often difficult. The leopard-spot distribution of the signs of consolidation in both the lungs, the rapid breathing and a highly toxic picture point to bronchopneumonia. I have nearly always observed the patient puffing, especially when the temperature is 102°F. or above. The grunt begins with each expiration and the pause that follows is absent. The dyspnœa is magnified by this grunt and the pause following each inspiration. Sucking in of the intercostal spaces indicates diminution of normal lung tissues and is often met with in moderate and severe cases.

The same type of grunt and dyspnœa is also present in lobar pneumonia, though the latter is less often seen in children. When it does occur, the toxæmia is less severe in lobar pneumonia but the accompanying cyanosis is well-marked. Physical examination usually reveals the lobar distribution of the pneumonia except when it is deep-seated and central. Herpes labialis is a common feature in lobar pneumonia. Sucking in of the intercostal spaces is absent in lobar pneumonia even when the dyspnœa is quite severe, though the affected side of the chest moves less during respiration. Lobar pneumonia

* Specially contributed to THE ANTISEPTIC.

clears up by *crisis*, unlike the broncho-pneumonia which tends to clear up by *lysis*.

Dyspnœa rarely occurs in cases of bronchitis even when other symptoms and signs are quite severe. Early cases of broncho-pneumonia closely resemble this condition and an accurate differentiation between the two is really difficult to make at this stage. Cases starting in bronchitis often end in bronchopneumonia, particularly when it is neglected in the early stages. In simple uncomplicated bronchitis, rhonchial fremitus is well felt and loud-moist rales are heard all over the lungs, the type of the breath sounds often being masked by the rales. No patches of consolidation could be made out.

When cases of asthma are complicated by bronchitis or broncho-pneumonia the signs are different. The dyspnœa is very marked and the rate of respiration is increased to twice or even thrice the normal rate. The wheezing that is present is mixed with loud moist rales. It is in this condition that the sucking in of the intercostal spaces is quite apparent and often confuses the physician.

In simple and pure asthma the signs and type of breathing in children are the same as seen in the adult except that in children the dyspnœa is likely to be less embarrassing to the patient. This dyspnœa is less orthopnoic and the child patient may often be seen to sleep quietly in spite of the dyspnœa.

Dyspnœa is one of the danger signals, when it is due to diphtheria of the fauces or larynx. When a white patch is present in the tonsils or fauces a smear of the material from the throat, examined under the microscope will reveal the cause. Since early diagnosis of this disease is essential, one cannot afford to wait or allow the dyspnœa to develop, and so the differential diagnosis must be borne well in mind. The common conditions which confound the issue are follicular tonsillitis and Vincent's infection of the throat. Neglect to examine the throat of the patient has cost many lives and so it is most important to examine as a rule the throats of all children with fever, cough or dyspnœa. Dyspnœa is usually ushered in when the patches in the fauces and tonsils have spread further down to the larynx. Unfortunately when the dyspnœa is due to pure laryngeal diphtheria, the finding is very vague and difficult to make, and the obstruction is quite severe. There may be nothing but dyspnœa or a croupy cough or a hoarse voice. When dyspnœa is present in this condition, sucking in of the intercostal spaces is invariably present. A swab from the pharynx often helps but cannot always be depended upon. When a case of bronchitis or bronchopneumonia with some toxicity and dyspnœa suggesting obstruction presents itself, a careful observation of the pulse and the picture of toxæmia, will help in recognising the malady. As a rule the temperature in uncomplicated diphtheria is rarely above 101°F.

Congenital syphilis affecting the pharynx and larynx may lead on to dyspnoea. The family history and other co-existing signs of congenital syphilis etc., should help to arrive at the diagnosis. Later, the blood may be examined for the Kahn and Wassermann reactions.

When dyspnoea is due to a foreign body, unless the history is confirmatory, an immediate laryngoscopy or bronchoscopy by a specialist is the best thing to do, though a laryngo-fissure is recommended in cases where the obstruction is above the level of the vocal cords. The dyspnoea is usually sudden and almost of a choking nature and the child is very restless.

A disease which is often missed is the acute retropharyngeal abscess. A good number of such cases are often dubbed as laryngeal diphtheria and a tracheotomy fails to save the patient. It is well to remember this and all that may be required is just a palpation of the back of the pharynx with the index finger in the throat and a fluctuant swelling situated to one side settles the diagnosis. Dysphagia and dribbling of saliva from the mouth are present. The head is fixed in a peculiar position and the glands at the angle of the jaw are enlarged on the affected side. There is an inspiratory stridor along with the dyspnoea.

There is a general belief that pulmonary tuberculosis is rare in children. This disease is as common in children as in adults. Whenever a lobar or bronchopneumonia does not clear up with the usual treatment tuberculosis must be suspected. A number of undiagnosed fevers with dyspnoea eventually turn out to be cases of miliary tuberculosis. When dyspnoea occurs in this disease, the damage is beyond repair. Sputum is rarely available and so a skiagram of the lungs is absolutely essential. It is not uncommon to see a case of pleural effusion or empyema with dyspnoea demanding immediate relief.

The dyspnoea that occurs in whooping cough is due to paroxysmal spasms of the respiratory passages. Clinically there is nothing to be relied upon, but when the coughing of the patient is heard, the physician may be sure of the condition. This disease occurs in epidemics and the presence of cases in the vicinity should help to surmise the cause. Early cases do not develop the whoop and so by hearing the characteristic cough alone, one should commence the treatment. Often a small ulcer is present over the frenum of the tongue. When secondary bronchopneumonia or bronchitis complicates, the dyspnoea becomes continuous.

When we turn to the cardiac conditions and diseases of the blood which give rise to dyspnoea, we come to the special type of dyspnoea which occurs on exertion. This develops when the cardiac compensation fails and results in a continuous dyspnoea as the end draws near. In this group, congenital heart diseases, mitral disease of the heart and failure due to anæmia, especially that resulting from

ankylostomiasis are the chief ones. Examination of the heart and blood reveals the underlying cause. The dyspnoea due to heart-failure is orthopnoic in character whereas in anæmic conditions the orthopnoea is not marked; the patient may even prefer to lie down. Infants rarely suffer from this group of diseases.

I wish to emphasize that in treating a child with dyspnoea, a careful search should promptly be made to find out the cause, since any delay in diagnosis will cost the life of the patient. Too much reliance on the modern antibiotics empirically administered without sufficient indication will only hasten the end of the patient.

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Fumagillin in Amœbiasis

This antibiotic isolated by Hanson and Eble in 1949, was first described as an antiphage agent. It occurs in crystals and is reported to contain only C.H. and O. It has shown only slight, antibacterial or antifungal activity. Its chief attribute as a direct-acting amœbicide was demonstrated in 1951 by McCowen *et al.* (*Science*, 113: pp. 202-203). Anderson, and his coworkers of the University of California and the American University of Beirut in Lebanon, studied the therapeutic value of fumagillin in chronic amœbiasis of macaques and man.

On the basis of preliminary observations in monkeys and the findings in other animals which were noted in the Upjohn Laboratories, cautious trials were begun on 20 chronically infected patients in Beirut. [Note: Three uninfected volunteers in the laboratory took the doses indicated before the trial on patients]. Single doses of 5 to 10 mg. per day for a total dosage of 50 to 100 mg. were given orally over 12½ days. There were no significant changes in blood urea nitrogen, serum bilirubin, nor in prothrombin test levels. As ambulant patients were treated, larger doses were not used. No untoward side effects were noticed.

In order to appraise fumagillin critically, a control group of ten untreated patients (cyst passers) was observed over 3 or more months and two other groups of 18 patients in each were given thiocarbasone and terramycin respectively, as Anderson *et al.* had found this to be the most effective amœbicides.

Summary of results:—Fumagillin orally is active directly against the *E. histolytica* in macaques and man. In man, at a total dosage of 100 mg. over 12½ days 9 out of 10 cyst-passers remained clear during 3 months. 50 mg. cleared only 4 of 10 patients. A comparative study with untreated controls and with terramycin and thiocarbasone treated patients was also made. *Fumagillin was most potent and appeared to be most active in chronic drug resistant intestinal amœbiasis.*—(*Am. Jour. Trop. Med. and Hyg.*, 1: 4, July 1952, pp. 552-558).

HYSTERICAL MANIFESTATIONS*

(A Critical Study of Some Aspects)

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(Continued from page 117 of Feb, 1953 issue of the 'Antiseptic')

PARALYSIS of the larynx is common in hysteria producing aphonia—which may sometimes clear up suddenly and dramatically. It is always the movements of the adductors that are affected—so that the cords are widely separated and cannot voluntarily be brought together, although they move freely during respiration and coughing when these acts are automatically performed without the intervention of the will. In the distribution of the cranial nerves—laryngeal and rarely facial paralysis are the only conditions met with. Squints and ptosis that are occasionally seen are due to spasms.

Tremors may occur, ranging from the finest to the coarsest movements, varying also from time to time, as in all manifestations of hysteria. They do not resemble tremors observed in organic disease. Localised hysterical spasms e.g. of the eye muscles, are of common occurrence; the tongue may also be rarely affected. Spasmodic ptosis and convergent strabismus may also occur. This ptosis which sometimes simulates a paralytic condition is characterized by the spasm of orbicularis palpebrae without any compensatory contraction of the occipito-frontalis—as in the paralytic form. Rarely the hysterical ptosis may be due to relaxation of the levator palpebrae.

Hysterical rigidity and contractures of the limbs are fairly common, rigidity being the outstanding feature in our cases. Organic rigidity and contractures can be overcome; but hysterical rigidity in its extreme form cannot be easily manipulated. A young woman used to get into bilateral rigidity of the lower limbs and she got very much worse whenever the husband visited her. Our endeavours to overcome them by force, only increased the opposition. The limbs, however, got relaxed during sleep. Anaesthetics also cause relaxation of such rigidity and contractures, that cause pain. Localized contractures occur in the feet, knees and hips. A cataleptic condition may also occur in which there is a loss of voluntary motion and a peculiar plastic rigidity of the muscles, as a result of which the limb may retain for an indefinite time any position in which it is placed.

* Specially contributed to THE ANTISEPTIC.

Spasms of the respiratory muscles may also occur with hysterical cough, hiccough, yawning and globus. Hysterical barking is characteristic and dyspnoea and hyperapnoea are common. Oesophageal spasm producing dysphagia simulating organic stricture, is frequently seen. Convulsions resembling epileptic fits are exceedingly common, and are generally ushered in by some hysterical sensation—as the globus hystericus, flatulence, or palpitation. Generalized tonic spasms then occur and the patient falls or slides down to the ground; but not with the sudden helplessness characteristic of epileptic fits. The tonic stage may be succeeded by clonic movements which are often very wild and uncontrollable. In severe cases opisthotonic and other elaborate complex postures of the body are assumed. The patient may scream during convulsions. Corneal reflexes are never lost and incontinence does not occur. After a variable time which is usually much longer than in epilepsy, the movements cease, as if the patient is tired. The normal mental condition then returns gradually. The most important consideration is to differentiate such hysterical fits from epilepsy. Very often they are indistinguishable and we usually rely on such small points as relaxation of the sphincters, injury to the body, biting of the tongue, character of the fall, the struggling form of the fits and so on. As the two conditions resemble each other very closely in all other respects, we have ventured to suggest that hysteria belongs to the same common pool of the epilepsies.

An extreme and interesting condition of hysterical contracture and spasm is illustrated by the hysterical type of non-gaseous abdominal bloating. Alvarez recently reviewed 92 such cases. The syndrome is similar to that described in the past under various terms *e.g.*, "phantom tumour" "pseudo-ileus" or "accordion abdomen." There are several varieties of this syndrome, some severe, painful and disabling, and others in which it is mild, or occasional or only incidental to more serious troubles. 85 of the 92 cases reviewed by Alvarez were women. The main point of interest in these cases is that the pronounced bloating is due *not* to any excess of gas in the digestive tract but apparently to a contraction of the muscles lining the back and the upper end of the abdominal cavity. Sometimes there may be in addition a relaxation of the anterior abdominal wall. These changes often associated a pronounced lordotic posture tend to throw the abdominal contents forward and somewhat downward towards the brim of the pelvis; the swelling therefore, usually increases gradually during the afternoon and decreases at night, without the passage of any flatus. Nearly all the 92 cases reviewed by Alvarez were nervous, unhappy, neurotic or psychopathic. Many were relatives of insane persons or of persons who suffered from epilepsy, migraine or diabetes. The bloating was often found to start after excitement, annoyance, fright or fatigue. This is obviously a hysterical manifestation—as all other organic causes were excluded—even by laparotomy.

Anorexia nervosa is a most dramatic form of anorexia of psychoneurotic origin. Vomiting may be often hysterical. In Hurst's opinion, the nausea and vomiting of pregnancy is always hysterical unless associated with eclampsia or acute yellow atrophy of the liver. It is a protest against pregnancy on the part of the subconscious mind! On the sensory side anæsthesia is the symptom most frequently complained of as a hysterical manifestation. The variability of its distribution and intensity is the clue to its diagnosis. Further, there is no organic basis for such a condition. The most frequent distributions are over one half of the body, the distal portion of a limb, or a segment of a limb—or it may be in irregular patches; such hemianæsthesia—the loss to all kinds of sensibility may be complete or partial; or it may be a dissociated sensation. When it affects one half of the body, not merely the skin but also the mucous membranes on that side become anæsthetic. As stated above, the peculiarity of such anæsthesia is that it can be modified by suggestion. The anæsthesia may disappear during sleep. A peculiar phenomenon is sometimes observed in which a touch on one limb is felt on the opposite limb. This is known as *allocheiria*. Hyperæsthesia is the other sensory manifestation of hysteria. In such cases the sensibility is abnormally acute over circumscribed areas, pain and tenderness being the most prominent symptoms. There may be tender spots—the so called *hysterogenetic spots*; pressure on which may start even convulsions. Such spots are commonly seen over various vertebræ, below the mammæ, and in women over the ovarian region. Varieties of pain will simulate rheumatism, migraine and angina. Headache is common and may be so severe as to mimic that of brain tumour.

We have already referred to the motor disturbances of the ocular muscles. There may be sensory disturbances as well. The field of vision is frequently contracted in a manner suggestive of hysteria. There is constriction of both fields, but more so of one than the other, this being greatest on the anæsthetic side. This is known as *crossed amblyopia*. The perimetric vision becomes progressively contracted. Sometimes there may be disturbances of colour vision in which everything appears uniformly grey. Such a condition is known as *achromatopsia*. Short of this, the field for red is the last to be lost; whereas in organic disease it usually fails first. Hysterical hemianopia or complete amblyopia are very rare. Anosmia may be bilateral. Deafness and loss of taste may be present on the anæsthetic side.

Vasomotor disorders such as flushing, erythematous eruptions, local oedema may occasionally occur. Dermatographism has been recorded. Hyperpyrexia has been recorded in hysteria, but that probably was artificially induced. Coming to the mental side of the picture, the hysterics are very often emotionally unbalanced. Capriciousness, quick changes of moods, whimsical likes and dislikes are

evident in their mental make up. Amnesia or complete blankness in the continuity of memory is also often seen. With such endless variations in its manifestations—the diagnosis of hysteria is often difficult; and all attempts to get at the root cause may be baffling. To the experienced observer however, the diagnosis of hysteria is almost a matter of instinct. A very characteristic feature of hysteria which helps in diagnosis is that there is an attitude of unconcern with regard to the symptoms and the physical disability, as an otherwise normal person should be. The other mental attitude is an absence of frankness on the part of the patient. She is suspicious.

As regards the motor disturbances a careful examination will show that it does not strictly conform to the signs and symptoms of any known organic disease. Irregular and jerky attempts at voluntary movements are very characteristic of functional weakness. There is a general tendency in a hysteric for the reflexes to be exaggerated. Exaggerated tendon reflexes with normal or brisk abdominal reflex is particularly useful to exclude organic disease in cases of spastic hysterical paralysis.

TREATMENT:—Remembering the fact that the hysterical manifestations give subconscious satisfaction, because it has enabled the patient to evade some difficulty or to achieve some selfish motive, the treatment should be guided with a firm though sympathetic attitude. Again, as the symptoms were produced by a process of suggestion, the patient's suggestibility will often enable the symptoms to be cured by the same means. The patient should be removed away from all sympathetic, and fussy environments, thereby depriving her of her subconscious satisfaction in her disability which is a characteristic mental attitude in hysterics. She should be made to feel her inferiority so that her own personality will assert and suggest the cure; and suggestibility is a double-edged weapon in hysteria. The disability can be brought on and can be cured by the same process. It is not our intention to enter into the details of treatment. Psycho-analysis and Weir-Mitchell treatment, with complete bed-rest, total restriction of visitors except the doctor, disallowing letters and reading of books etc. should be instituted. Such a firm attitude in dealing with the patient and strict restrictions as stated above, will lead to ultimate success, though at first non-cooperation and even defiance by the patient may have to be encountered.

Prevention lies in properly educating the children to make their own decisions and to face up to difficulties. Under no circumstances should they be allowed to feel that they could have their own way by resorting to persistent defiance and fussing about.

A rational and reasonably sympathetic but firm attitude on the part of parents in bringing up their children, exercising kindly discipline at the same time, will go a long way in eradicating the germ of hysteria from them.

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Premenstrual Tension Associated with Psychotic Episodes

Williams and Weeks of the Howard Medical School New Jersey, discuss in a preliminary report sixteen cases of pre-menstrual tension associated with psychotic episodes. The symptoms included minor somatic complaints, followed by sudden changes in behaviour with either marked withdrawal or marked psychosomatic activity, hallucinations, and delusions; insomnia, increase in weight and low to normal blood pressure; the psychotic episodes were characteristic of either the manic form of manic-depressive psychosis or the catatonic form of schizophrenia. In some cases catatonic features occurred at the onset of menses at one time, mania at the other; in only 5 cases a total cessation of all symptoms occurred between menstrual periods.

Treatment varied. In 5 cases shock therapy was used during menstruation in the psychotic episode, followed by the use of ammonium chloride, (45 grains) daily for 5 days; in 2 cases liver extract 10 U.S.P. units was given for 3 doses, at intervals of 3 days. When shock therapy was used, the psychotic symptoms during the intermenstrual period were far less severe, and in most cases, completely subsided, only to return at the next menstrual period. Other treatments included shock therapy, *plus* a salt-free diet during psychotic episodes and psychotherapy during intermenstrual periods; shock therapy, ammonium chloride, psychotherapy, salt-free diet *plus* thiamine chloride; shock therapy, nicotinic acid, thiamine chloride, and progesterol prior to the onset of menses; coramine, calcium gluconate and salt-free diet; ephedrine sulphate, thiamine chloride and a salt-free diet prior to onset of menses. The longest recurrence of symptoms monthly was 7 to 9 months. In all others, the condition was brought under control by the third or fourth month.

It seems likely that the disorder has an endocrine basis, particularly an excess of oestrogens and a deficiency of progesterone, with associated water retention,—(*Jour. Nerv. and Ment. Dis.*, 116 : 321-329, Oct. 1952).

HEPATIC AMŒBIASIS*

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THIS term has been deliberately chosen, for the alternative term 'Amœbic Hepatitis' does not convey the entire pathology of amœbic infestations of the liver. Much has been written on the subject, while research has contributed its share to a better understanding of the problem and new curative drugs have been added to our armamentarium, but still the picture is not complete; the pathology has not been fully elucidated and complete cures are not obtained in all cases, relapses being still frequent.

Amœbic dysentery is endemic in Bharatpur because of its unsafe water supply. Drinking water is obtained from wells situated along a moat, running round the fort in the centre of the town. The water in the moat is stagnant and grossly polluted. The wells are situated very near the moat so that there is not adequate and efficient filtration during percolation. The town has not got a filtered protected piped supply. It is my firm belief that the town water supply from contaminated wells harbours *E. histolytica* and accounts for the endemicity of the disease in this area.

It is common knowledge that the active amœbæ reach the liver from dysenteric ulcers in the colon through the portal system, but whether lymphatics also serve as a channel for transmission is not definitely known. Having reached the liver they affect the liver cells, more so if they are devitalised by alcoholic excesses or nutritional deficiencies.

Clinically however, at this stage of invasion the patient does not suffer from any fever or manifest any signs in symptoms. The attack may be self-limited but with the possibility of flaring up later into activity. However, if the amœbic invasion is successful, the liver gets enlarged and tender in due course of time and the case presents certain signs and symptoms. This is a prelude to the ultimate formation of a liver abscess, in some, though not in all cases.

The patient complains of vague pains and heaviness in the right hypochondrium, rigors or chills with or without fever, arthralgia and body-ache, loss of appetite and distaste for food, disinclination for work, drowsiness or lethargy, sweating and bad taste in the mouth. These significantly point to some pathological condition in the liver and may be the result of destruction of liver cells and consequent impaired functioning. But then, the liver cannot be impaired so easily, particularly when its reserve power and potentiality for regeneration are indeed great. What then are the digestive symptoms due to? My own views on this point are:—

* Specially contributed to THE ANTISEPTIC.

1. It may be a liver in a state of shock, suspended animation and impaired efficiency due to a 'sit-down' strike of the liver cells.

2. It may be due to a destruction of the liver cells resulting in what may really be a protein-shock reaction, that accounts for the several somatic manifestations.

3. It may be due to a sub-clinical deficiency of vitamin B complex, the manufacture of which is partly or wholly impaired due to an altered environments created by a change in the bacterial flora of an inflamed intestinal tract. Vitamin B assay might be useful to clinch this issue.

4. It may be an affection of the gall bladder ; the amœbæ causing a sub-clinical cholecystitis, the symptoms of which are nearly the same. The wide prevalence of chronic cholecystitis in Bharatpur lends support to this hypothesis.

5. It may be due merely to mechanical factors resulting from enlargement of the liver which may press on the stomach and duodenum.

If this condition is not treated promptly and properly, it progresses to the next stage of abscess formation. It is believed that colliquative necrosis of the liver cells occurs in one or more centres and by confluence results in the formation of an abscess. The 'chocolate-coloured-pus' inside is only liquefied liver and is hence sterile, until and unless secondary infection takes place. Amœbæ are not as a rule found in this pus but only in the scrapings from the wall of the abscess cavity. Cysts are never found in the pus either. When it is secondarily infected, the colour and odour of the pus changes-particularly in *B. coli* infection. Emetine injections cut short the fever and this lends support to the view that the drug kills the amœbæ and arrests the necrosis. But I had a case where the temperature subsided, when the abscess burst into the pleural cavity even when no amœbicidal drug had been administered. This was perhaps due to a release of the tension.

The complications of liver abscess are very interesting. Pleural effusion consequent upon liver-enlargement is fairly common, but the why of it is still obscure. It may be due, they say, to mechanical pressure, allergy or some other 'reaction.' What this 'reaction' means is not clear. The abscess bursts into the surrounding organs most often into the pleural cavity and lung giving rise to stained sputum and a mistaken diagnosis of T.B. of the lungs. But pulmonary amœbiasis does occur without this bursting ; it is not known whether it is an extension from the liver or directly from the intestines. In one case chocolate stained serous fluid was aspirated from the space between the diaphragm and the liver. Segmental collapse or consolidation of a part of the right lower lobe of the lung has been seen, the ætiology of which again is difficult to explain.

Jaundice as a complication is rare and cirrhosis as a sequela is uncertain. And lastly, mental conditions like psychoses, neurosis, neurasthenia, melancholia and lack of concentration have been met with in mixed infections of hepatic and intestinal amoebiasis. Whether they are due to vitamin B deficiency or to an impaired detoxicating function of the liver or due to cerebral parasitosis, it is difficult to say.

TREATMENT should be in two stages. For the initial stage of 'hepatitis' (with fever and an enlarged tender liver), the emetine and chloroquine groups of drugs are quite effective. The temperature quickly subsides, the tenderness of the liver disappears and the enlargement gradually comes down; but the digestive symptoms are not so easily shed by this treatment. The simultaneous administration of both emetine and chloroquine has no particular advantage, and may perhaps be undesirable too.

Case Reports.—CASE 1.—An elderly man, of about 45 years, had low evening rise of temperature and cough for about 2 months. He was treated by local practitioners with antibiotics and antitubercular drugs without any effect on the temperature. Slight enlargement and tenderness of the liver raised my suspicion for liver parasitosis; and a skiagram showed clear lungs but raised and deformed right dome of the diaphragm. He was then put on Nivaquine and the temperature was controlled within 3 days.

CASE 2.—A woman about 35 years of age, was running irregular intermittent temperature for about a month. Clinically nothing abnormal was detected, except an enlarged tender liver. Her lungs were X-rayed with negative result for tuberculosis. Laboratory findings did not reveal anything positive. She was given 6 emetine injections; the temperature promptly came down and has remained normal ever since.

CASE 3.—A girl, aged 16 years, had bouts of irregular temperature with rigors at intervals of about a week. The spleen was not palpable, liver slightly enlarged and tender; M.P. not found in blood. No history of dysentery. She had been treated with anti-malarial drugs for about a month without avail. Clinically it was diagnosed as a case of 'amoebic hepatitis' and a course of Nivaquine cured her.

CASE 4.—An adult, about 22 years of age, was running temperature with rigors for about a fortnight. Complained of fullness after meals, disinclination for work and felt depressed. Liver slightly enlarged. E.H. present microscopically in the stools. He was put on Camoquine and was cured.

The treatment of a full-fledged liver abscess is not so satisfactory, in the sense that no medicine seems to clear up completely the 'pus' that collects in the liver, though fever and other manifestations are easily brought under control. Aspiration has very often to be

resorted to, whenever the collection is sufficiently large and may have to be repeated often. The procedure of exploring and aspirating the liver has to be blind and empirical and consequently difficult and incomplete in some cases.

My own views on the treatment of liver abscess can be summed up thus:—

The chloroquine group of drugs and emetine are useful in combating the fever and associated symptoms. Aspiration becomes essential in most cases, and open drainage of the liver has proved to be unsatisfactory. Injections of emetine into the abscess cavity itself, which were formerly in common use have now been practically discarded as useless. Aureomycin and terramycin have little or no place in the treatment of hepatic parasitosis, though they have been very useful in amœbic dysentery. The toxicity of chloroquine is low and the margin of safety is fairly wide.

Here are a few interesting case records:—

CASE 1.—An old emaciated man of 45 was running an evening temperature and complained of cough. A skiagram of his lungs was indefinite for T.B., and his doctor put him on anti-tubercular treatment. His condition became worse and the temperature persisted; he developed œdema and anæmia. One day he felt severe stabbing pain in his right chest and presented a picture of collapse. His temperature suddenly fell to subnormal. I was consulted at this stage and I diagnosed it as a liver abscess which had burst into the pleural cavity. He was X-rayed again and the fluid level was found to be upto the second rib anteriorly. He was aspirated and a kidney-tray full of typical 'anchovy sauce' pus was removed. His temperature remained normal since the day of collapse due to the bursting of the abscess and remained so without any further medication. As he was very weak and emaciated, emetine was not considered advisable and a course of Nivaquine was started instead. He had to be aspirated again after a week and his progress was satisfactory. He was then seen by another doctor who advised blood transfusion; but the man died a day after the transfusion. The interesting point is that the temperature came down and remained normal after the abscess had burst into the pleural cavity.

CASE 2.—A woman, aged 22, complained of pain in the liver area, fever with rigor and diarrhœa for about 2 months. Liver was found enlarged and tender, stools negative for E.H. on microscopic examination. She was put on a course of Nivaquine which controlled the temperature, but not the pain. After a fortnight she had temperature again, and the pain in the liver area increased. She was given a course of emetine. The temperature came down to normal but the pain had been only slightly reduced. After sometime she had a recurrence of the trouble. She was aspirated

and 100 c.c. of typical pus removed. Her complaint appeared to have vanished but only to recur after 3 weeks. She was again running a temperature. Again she was aspirated and put on penicillin injections and Nivaquine orally. Her condition continued to deteriorate and ultimately the Surgeon decided to effect an open drainage. Since then she maintained irregular temperature; the liver continued to increase in size, became hard and cirrhotic. Finally she left the hospital in a very poor state of health.

CASE 3.—A boy aged 18, came with a tender lump in the epigastrium and a daily hectic rise of temperature. He was diagnosed as a case of amoebic abscess of the left lobe of liver and was put on emetine injections and Nivaquine orally. His temperature came under control within 3 days, but the pain and lump subsided only partially even when the course of the amoebicidal drugs was finished. Aspiration was therefore, tried but without success—(the cause of failure being attributable to the axillary approach instead of through the abdomen). His pain persisted till he left the hospital; and I was informed later that the abscess burst through the anterior abdominal wall. This shows the difficulty in aspirating liver abscesses of the left lobe of the liver even by experienced surgeons.

I am thankful to the Principal Medical Officer, Bharatpur, for his permission to send this article for publication and to Messrs. May & Baker for a supply of *Nivaquine* and to Messrs. Parke Davis for *Camoquine*.

Medical Treatment of Renal Tuberculosis

Chauvin *et al.*, treated 100 patients with tuberculosis of the kidneys using a special scheme of their own:—Each patient was given more than two or three courses of treatment lasting for 30 days, followed every time by a 30 days' rest between every two courses. Each course consisted of two stages; during the first 10 days, the patient was given intramuscular injections of 5 c.c. of chaulmoogra oil every other day. During the second period of 20 days, the patient received daily intramuscular injections of 2 c.c. chaulmoogra oil, 2 gm. of streptomycin intramuscularly in 2 divided doses, and 7 to 14 gm. of PAS orally in gradually increasing doses. This continuous treatment caused little fatigue to the patient and did not seem to induce streptomycin-resistance, since functional signs of improvement manifested themselves mainly within the first few days of the second period. The results of the treatment should be evaluated only after three courses.

Only 24 of the 100 patients were given three courses and were observed for more than a year. Of these 24, nine apparently recovered with disappearance of functional symptoms as well as of pus and Koch's bacilli from the urine. Eight were greatly improved with regard to general condition and cystitis; but pus and bacilli were still present in the urine. The remaining seven were therapeutic failures. This scheme of treatment, say the authors, may be expected to give relief to patients with early tubercular lesions of the kidneys in the absence of pronounced changes in the ureters.—(*Press. Med.*, 59: pp. 1281-1300, 1952).

ACCIDENTAL POISONING IN CHILDREN*

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CHILDREN are likely to be victims of accidental poisoning as a result of their own activities or of the wrong administration by careless elders. The young ones mistake the poisons for eatables and put them into their mouths, particularly when solid in shape, resembling edible fruits or seeds. Again as part of playful indulgence, the little ones may transfer anything (solids, or liquids, or pastes or bottled stuff) into the mouth too readily.

I wish to record in this communication, my observations on accidental poisoning in a few children who came under my care in the Government Royapettah Hospital, Madras.

1. Kerosene oil poisoning: two cases. 2. Yellow oleander poisoning (*Cerbera thevetia*, N. O. Apocyanaceæ): one case. 3. Potassium permanganate poisoning: one case. 4. Linimentum terebinthinæ poisoning: one case. 5. Physic nuts poisoning: (*Jatropha curcas*, N. O. Euphorbiaceæ): four cases. 6. Concentrated nitric acid poisoning: one case.

Kerosene oil poisoning is very common. Kerosene is one of the liquid hydrocarbon derivatives got by fractional distillation of petroleum.

The symptoms observed by me were:—Fever, bronchopneumonia, abdominal distention, drowsiness and convulsions. Termination is sometimes fatal. Steiner noticed cerebral (drowsiness), pulmonary (pneumonia) and degenerative signs (cardiac and renal damage with cellular casts, hepatosplenomegaly and occult blood in motions) in children with kerosene poisoning; and further suggested gastric lavage with weak sodium bicarbonate solution, and use of cardiac stimulants, antibiotics, and oxygen against pulmonary oedema and infection. Bologna and Woody stress the prominence of pulmonary and nervous symptoms and warn against the ever present danger of pulmonary oedema which they tackled with early gastric lavage and use of antibiotics and oxygen therapy. A late result which has been stressed, is bronchiectasis which is prevented by hyperventilation and postural drainage.

In the case reports discussed below the writer has experienced bronchopneumonia and drowsiness as also persistent tachycardia. Convulsions were experienced in other instances not narrated here.

1. A male child 1½ years old, in good nutrition was admitted on 16-1-50. The child came into the hospital long after the poisoning; and after stomach wash, the child was put to bed; and even thereafter developed signs and symptoms of bronchopneumonia, pyrexia and drowsiness. Previous history: nil particular. Breath

* Specially contributed to THE ANTISEPTIC.

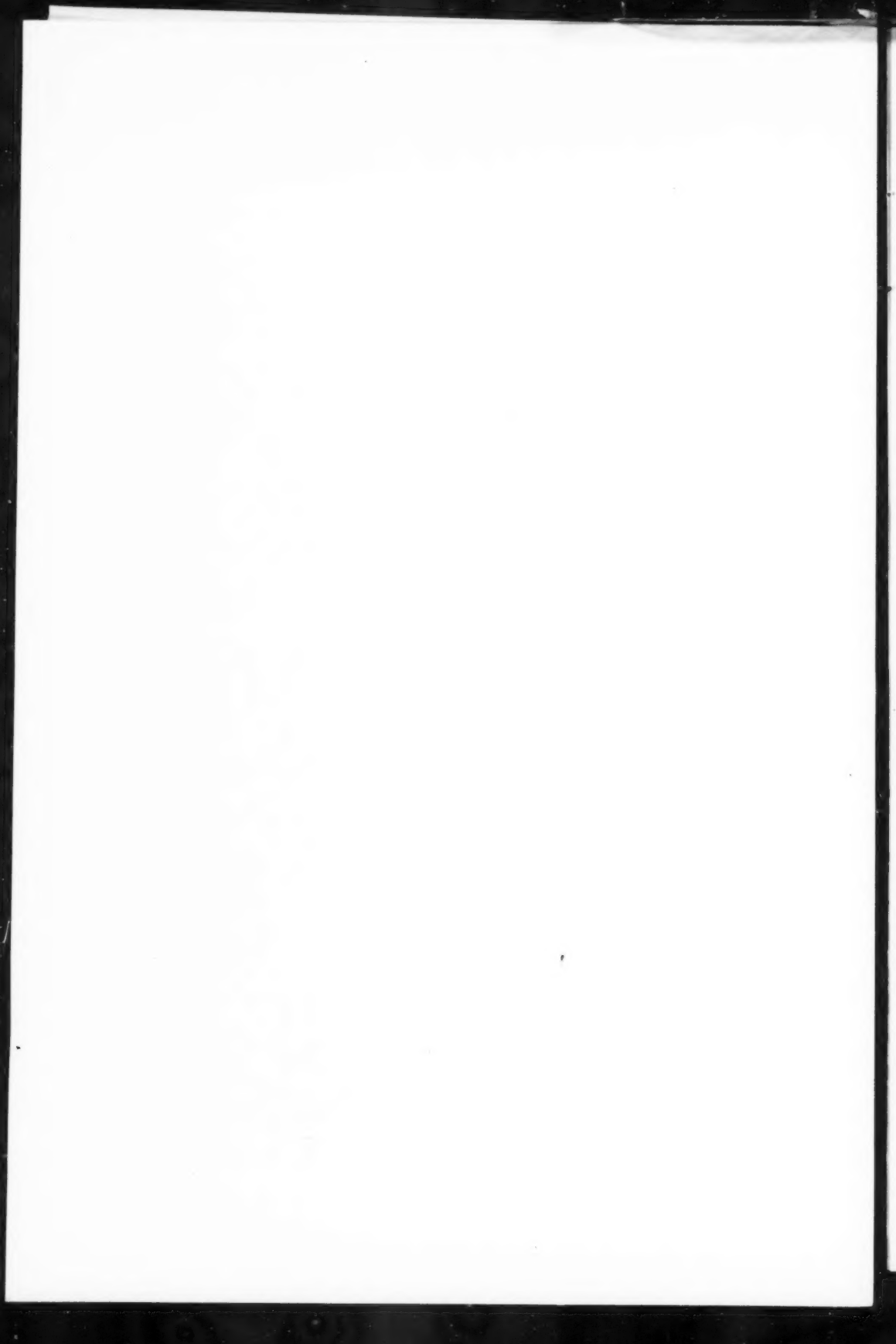
Accidental Poisoning in Children

A. V. S. Sarma.



X-rays chest A.P.—Kerosene Oil Poisoning
—Left basal opacity and diffuse lung mottlings
seen.

[Vide page 197.



smelt of kerosene. Drowsy. Abdomen tumid. Liver hard and felt 1" below costal margin. Spleen not palpable. Lungs: breath sounds harsh over the bases with tachypnoea. Heart sounds rapid and clear. Blood: No malarial parasites seen. Urine: No sugar and no albumin. X-rays chest taken when the child could be moved about: Findings are marked basal shadows; and pneumonitis and pleuritis over the left base. Also diffuse mottling of lung fields seen. (*Vide* X-ray plate).

TREATMENT:—Stomach wash was given, as also coramine 1 c.c.; and penicillin 2 lacs per day given on 3rd, 4th, and 5th February 1950. A simple expectorant mixture containing Tr. Hyoscyami given orally with the expectation that Hyoscyamus would decrease pulmonary secretions. Temperature was particularly high (103°F.) on 4-2-'50. Discharged cured on 6-2-'50.

2. A female child 1 year old. Admitted on 10-4-'50 for accidental poisoning with kerosene; the baby having swallowed some oil from a kerosene lamp left on the ground. Nutrition average. Respiratory: Breathing rapid with expiratory grunt. Breath sounds harsh all over the chest with no rales. Heart: action rapid, regular but sounds tic tac. Abdomen—tumid. Liver and spleen—palpable.

Urine:—No sugar and No albumin. Blood: No parasites seen.

TREATMENT:—Emetic given on admission with good result. The rest as in case 1. Diet consisted of milk and white of one egg. Discharged cured on 14-4-1950.

COMMENT:—Kerosene oil is probably absorbed by the gut and it circulates in the blood stream and possibly gets excreted by the lungs and in this excretory phase, a broncho-pneumonia develops with respiratory distress and drowsiness and convulsions. This is my opinion, while a regurgitation into the respiratory tract may also occur as recorded by others.

Kerosene oil poisoning is a serious condition with cerebral depression and vagal paralysis with respiratory inhibition and therefore, always demands immediate as well as sustained attention.

Yellow oleander poisoning.—Yellow oleander poisoning can be intentional. In the reported case, the child was reported to have eaten a nut of the plant mistaking it for almond and thereafter vomited portions of the nut.

Child R., female, 4 years old. Admitted on 28-1-'50. Vomited twice after eating the nut and also purged once. At 1 A.M. she was put into the hospital. 1 c.c. of coramine subcutaneously was given as immediate stimulant.

Child in moderate nutrition. Eyes sunken. Tongue clean. Consciousness present. Pulse 140 per min. regular but volume and tension poor. Heart sounds tic tac. Abdomen soft, liver and spleen not palpable, Lungs: clear.

28-1-'50.—Diet: milk, tea, white of eggs.

TREATMENT:—Kaolin drams $1\frac{1}{2}$ with carbon dram 1; made into 3 powders—one t.i.d. Also coramine 1 c.c. by subcutaneous injection. Stimulant treatment continued. The child was discharged cured on 30 Jan. 1950.

COMMENT:—The toxic action is due to thevetin acting like digitalis. To overcome the poisoning, antidotes as for digitalis intoxication are helpful. E.C.G. of a child recovering from yellow oleander poisoning appears normal.

Potassium permanganate poisoning.—Potassium permanganate is a corrosive reported to produce abdominal pain and collapse as also cardiac paralysis.

Child V., female, 1 year old; admitted on 11-4-'50 for having swallowed accidentally an unknown quantity of potassium permanganate. Hands stained black. Child in good nutrition. Abdomen soft, liver and spleen both palpable. Lungs: breath sounds harsh over right upper zone with a few rales.

Urine:—No sugar and no albumin. Fæces: nil particular.

TREATMENT:—Coramine 1 c.c. subcutaneously given. Glycerine enema was given. Castor oil emulsion given internally. Diet: milk and white of egg. Discharged on 12-4-'50 (cured).

COMMENT:—The child had probably taken a very small quantity and therefore, no serious symptoms were encountered.

Manganese can damage the liver and the basal ganglia; and late results like cirrhosis of liver or extra-pyramidal rigidity are possible in serious cases. Knowledge would be welcome in this connection.

Linimentum Terebinthinæ poisoning.—Child J., male, 10 months' old, swallowed an unknown quantity of linimentum terebinthinæ. The bottle of the liniment was meant for the parent but served as a source of poisoning to the child. Admitted on 7-4-'50. The child was conscious. Abdomen soft. Liver 1 inch enlarged; and spleen just palpable. Temperature remained throughout normal. Heart and lungs: nil particular.

TREATMENT:—Sodii sulphas gr. 20 with Sodii bicarb gr. 15 with small quantity of water (2 dr.) given on admission. Also glycerine enema. 8-4-'50. A carminative mixture with sodii sulphas given orally. Also a glycerine enema. Diet: diluted milk and fruit juice. White of one egg. Discharged against medical advice on 8-4-'50.

COMMENT:—Oil of turpentine is viewed as renal irritant and as a possible ætiological agent of nephritis.

Physic nuts poisoning:—The seeds of the *Jatropha* plant resemble castor oil seeds and the oil expressed from the seeds has an action akin to the oil from the seeds of *croton tiglium* though milder in degree—severe diarrhœa and collapse are the dreaded results.

Four children went into a neighbouring garden to play and ate the seeds of *Jatropha curcas* and all of them had to be removed to the hospital for drowsiness and gastroenteritis.

Treatment was the same for all and consisted of a stomach wash, and stimulant treatment including 2 c.c. of coramine subcutaneously.

1. M., female, 8 years. Admitted on 1-12-'50 and discharged cured on 4-12-'50, ill-nourished, tachycardia present. Abdomen soft. Liver palpable. Cæcum felt thickened. Heart and lungs: nil particular. Pulse and respiration noted as below:—

Date	Time	Pulse rate	Respiration rate	Observation
1-12-'50	2 p.m.	126 per min.	28 per min.	Vomited a round worm.
	3 "	126 "	28 "	
	6 "	110 "	26 "	
	7 "	110 "	24 "	
	8 "	108 "	24 "	
	9 "	108 "	24 "	
	11 "	100 "	24 "	
2-12-'50	3 a.m.	98 "	24 "	
	4 "	98 "	24 "	
	5 "	94 "	24 "	

2. L., male, 9 years. Admitted on 1-12-'50 and discharged cured on 2-12-'50. Ill-nourished child with marked conjunctival xerosis. Abdomen soft, liver and spleen not palpable. Heart and lungs: nil particular. Pulse and respiration noted as below:—

Date	Time	Pulse rate	Respiration rate	Observation
1-12-'50	2 p.m.	140 per min.	28 "	
	3 "	140 "	28 "	
	6 "	104 "	24 "	
	7 "	100 "	24 "	
	8 "	102 "	24 "	
	9 "	102 "	24 "	
	11 "	102 "	24 "	
	12 night	102 "	24 "	
2-12-'50	3 a.m.	94 "	26 "	
	4 "	94 "	24 "	
	5 "	92 "	24 "	
	6 "	92 "	24 "	

3. S., female, 10 years old. Admitted on 1-12-'50, and discharged cured on 4-12-'50. Regained consciousness $\frac{1}{2}$ hr. after 2 c.c. of coramine was given by subcutaneous injection. Moderately well-nourished. Tongue clean. Abdomen soft; liver and spleen not palpable. Pulse and respiration noted as below:

Date	Time	Pulse rate	Respiration rate	Observation
1-12-'50	2 p.m.	120 per min.	23 per min.	Unconscious on admission. Vomiting and diarrhoea present.
	3 "	120 "	28 "	Consciousness returned.
	6 "	120 "	26 "	
	7 "	102 "	24 "	
	8 "	100 "	24 "	
2-12-'50	3-30 a.m.	86 "	24 "	
	4-30 "	86 "	24 "	
	5-30 "	84 "	22 "	

4. P., female, 12 years old. Admitted on 1-12-'50. Discharged cured on 4-12-'50. Moderately well-nourished. Abdomen soft; liver and spleen not palpable. Lungs: breathing rapid. Heart: tachycardia present on the first day.

All the four children had a diet of diluted milk, and white of eggs.

COMMENT.—Physic nuts are regarded as anthelmintics (Kirtikar and Basu). The oil from the seeds was studied (*Agric. Ledger*, 1911; *Jour. Soc. Chem. Ind.*, 1914). The active principle of the oil is jatrophic acid, but the seeds owe their toxic properties to a toxalbumin called curcin and analogous to ricin. One child under the influence of toxic action vomited a round worm, but whether this is due to mechanical reasons or an anthelmintic action of the ingested poison cannot be decided. At present santonin is a rare commodity and the writer feels that a safe anthelmintic, if obtainable from the physic nuts, would be welcome to the profession.

Concentrated nitric acid poisoning.—A girl of 1½ years is said to have swallowed concentrated nitric acid and was admitted (17-8-'51) with sore mouth and marks of acid on the lips and abdomen. Dysphagia, pyrexia and bronchitis with bronchopneumonia present. Rest milky feeds, alkaline powders and penicillin therapy saved the child.

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THIRD GENERATION SYPHILIS

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CAN syphilis be transmitted through three generations? Evidence produced by Fournier answers the question in the affirmative. Nabarro recorded seventeen families proving the passage of syphilis through three generations.

The difficulties in proving syphilis through three generations are obvious. A congenitally syphilitic parent may have an infected child, but the difficulty in excluding acquired syphilis in the other parent is very great.

The following information regarding two families shows that syphilis can pass on to the third generation.

(1) A female child aged $1\frac{1}{2}$ years was seen by me for enlargement of the liver. The child appeared apparently healthy but for a soft and painless enlargement of the liver and palpable spleen and a depressed bridge of the nose. The mother had an abortion a year prior to the birth of the child, and the Wassermann reaction of the mother's blood was positive. The father of the mother had treatment for gummata over the shins. The father of the child was examined, but no history of exposure or signs of infection could be detected.

The child improved remarkably well on grey powder (during pre-penicillin days), the liver enlargement simply melted away under the influence of mercury.

(2) A female child 3 months old, was seen by me for fits. The infant had a large head, and a liver which was felt near the umbilicus. A child born previously died of similar fits. The mother looked a typical congenital syphilitic with depressed bridge of the nose and deformed teeth and complained of headaches. Her blood gave a positive Wassermann reaction. A few months later, the mother had hemiplegia, and Wassermann reaction of the patient's blood was strongly positive. The husband of the congenitally syphilitic mother was examined by me, but no suspicion of syphilis was valid within the knowledge of the patient.

The child got convulsions and died later. The mother had a miscarriage during the course of the next year, and died of generalised convulsions and weakness.

The members studied in the above families were under my care during seven years, and were therefore, examined from the particular viewpoint under discussion.

Thus syphilis is shown to be capable of being transmitted through three consecutive generations.

A CASE OF STARVATION OSTEOMALACIA

M. G. VARADARAJAN M.B., B.S., D.M.B.,

AND

K. RADHAKRISHNAN, M.B., B.S., D.M.B.,

Radiologists, Erskine Hospital, Madurai.

Patient.—K., adult male, aged 25, native of Ramnad, beggar by profession was admitted into the skin-ward of Erskine Hospital, Madurai on 10-11-'50 for avitaminosis, pain of a dull boring type in the back and muscular weakness of one year's duration.

Appearance of patient:—Short in stature, marked kyphosis of the dorsal region reducing his height by several inches. There was no hair on the scalp. Skin dry, exfoliative dermatitis was present. Teeth normal. Patient had a waddling gait.

Examination of the central nervous, cardiovascular and respiratory systems revealed no definite abnormalities.

Investigations.—24-11-'50. Blood calcium 11.3 mg.; blood phosphorus 8.0 mg.; blood for Kahn negative. Total R.B.C. 3.2 million; T.W.B.C. 17,200; Hb. 62 per cent; P-45; E-31 per cent; L-20 per cent; and M-4 percent. Stools and urine examinations revealed no abnormality.

X-ray appearance:—14-11-'50.—The radiographs of the bones showed general osteoporosis and pressure deformities. The bones of the pelvis showed all the deformities one would expect to see in softened bone. General osteoporosis was seen. The sacrum was pushed down from its normal into an horizontal position. The bodies of the ilea were depressed and flattened laterally. The femoral heads pushed in the lateral walls of the pelvic canal and the brim became tri-radiate. A number of pseudo-fractures appeared to be visualised, extending only partially through the length of the body of the ileum, ischium and pubic bones. The rami of the pubic bones had been severed as a result of the crushing.

27-10-'50.—*Vertebral bodies* of the lower thoracic and lumbar especially were biconcave. The periphery of the bodies generally showed a greater density than the interior which showed a little trabeculation.

14-11-'50. *Skull:*—The basilar impression in the base of the skull due to the yielding of the base around the upper cervical vertebrae was not very marked. One faint small oval circumscribed area of decalcification was visualised in the lateral view of the skull.

23-10-'50 and 15-11-'50. *X-ray: chest and ribs:*—There was general osteoporosis of the ribs and pseudo-fractures were visualised in the scapulae, pseudo-or true fractures of ribs were well marked in 6, 7, 8 and 9th ribs of the right side and 2, 4, 5, 6, 9, 10th ribs of the left side. The intercostal spaces appeared to be narrowed.

A Case of Starvation Osteomalacia

M. G. Varadarajan and K. Radhakrishnan

Before Treatment



Pelvis. 14-11-50.—The bones of the pelvis show all the deformities one would expect to see in the softened bone (see the article for details)



27-10-50.—Lat. view of lumbar vertebrae and sacrum



23-10-50.—Base and cervical vertebrae
—Lateral view.

A



23-10-50.—Cervical bodies and ribs
(A. P. view).

A Case of Starvation Osteomalacia
M. G. Varadarajan and K. Radhakrishnan

Before Treatment



14-11-'50—Skull-lat. view. The basilar impression in the base of the skull due to the yielding of the base around the upper cervical vertebrae was not very marked. One faint small oral circumscribed area of decalcification was visualised in the lateral view of the skull.



11-12-'50—Right humerus. Bony trabeculations are not well visualised and pseudo-fractures are visualised in upper and middle thirds of shaft of right humerus.



11-12-'50.—Both bones of right forearm. Trabeculations are not well visualised and a pseudo-fracture in the middle third of shaft of right ulna is visualised.



15-11-'50.—Chest and ribs. Report—There was general osteoporosis of the ribs and pseudo-fractures were visualised in the scapulae, pseudo or true fractures of ribs were well marked in 6, 7, 8, 9th ribs of the right side and 2, 4, 6, 9, 10th ribs of the left side. The intercostal spaces appeared to be narrowed.

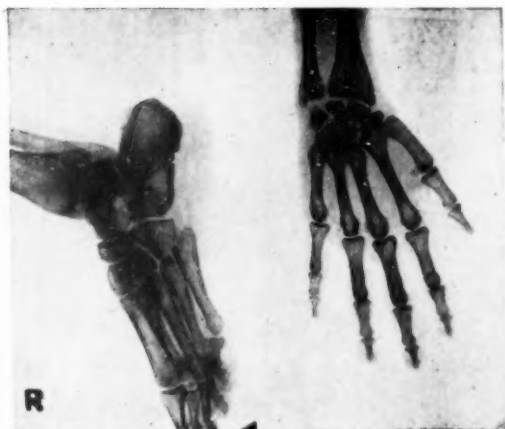


24-11-'50—No marked dilatation of the colon was visualised.

A Case of Starvation Osteomalacia

M. G. Varadarajan and K. Radhakrishnan

After Treatment



25-4-'52.—(Lateral view of foot). Rt and anteroposterior view of hand—Rt.



25-4-'52.—Both bones of right leg and right femur.



25-4-'52.—Lateral view of thoracic vertebrae.



25-4-'52.—Pelvis: Note the marked difference compared with the one taken on 14-11-'50 before treatment.

A Case of Starvation Osteomalacia

M. G. Varadarajan and K. Radhakrishnan

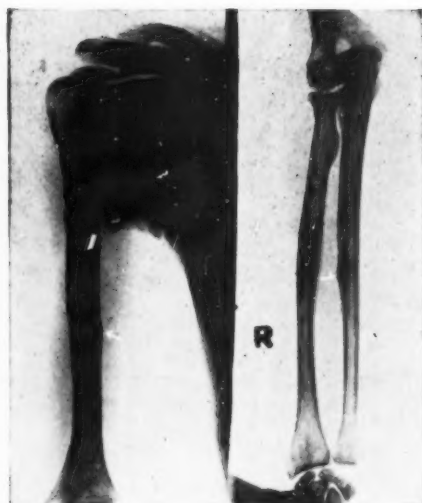
After Treatment



25-4-'52.—Lateral view of skull. Small oval circumscribed area of decalcification visualised in the picture taken on 14-11-'50 has disappeared.



26-4-'52.—Lateral view of lower thoracic and lumbar. Bodies of the vertebrae are assuming their normal shape due to treatment.



25-4-'52.—Right humerus and right radius and right ulna showing the changes after treatment. Pseudo-fractures have disappeared and their place is shown by slight thickening of the cortex.

11-12 '50. *The long bones*:—In both bones of the right forearm and in the right humerus bony trabeculations were not well visualised and pseudo-fractures were visualised well, especially in the shafts of the right humerus and the right ulna (upper and middle-third of the humerus and the middle third of the ulna).

These are called Looser's Zones. Looser describes that on microscopic examination "the old laminated bone disappears by lacunar absorption being replaced at the same time in the marrow by mottled bone devoid of calcium to begin with." They often appear without any trauma. There are however, arguments to support the view that they are caused by trauma. They are found in most of the bone diseases in which softening or bending occurs on the convex aspect of the curvature at the point where one would expect a bent article to fracture. They frequently do not extend to the whole width of the bone.

Barium enema:—No marked dilatation of the colon was visualised.

DIFFERENTIAL DIAGNOSIS:—(1) In hyperparathyroidism there is a high serum-calcium, a low plasma-phosphorus and an increased calcium excretion in the urine.

(2) In senile osteoporosis—the blood chemistry is normal and marked kyphosis is present.

(3) *Thyrotoxic osteoporosis*:—The usual signs of hyperthyroidism are present and blood chemistry is normal.

(4) In multiple myeloma Bence-Jones protein is found in the urine in 75 per cent of cases, the serum-globulin is usually increased and albumin-globulin ratio diminished. The serum-calcium is usually normal but sometimes raised. The plasma phosphorus is usually normal but rises in cases showing renal insufficiency. In radiographs, the condition may closely resemble osteomalacia.

(5) *Osteomalacia* in idiopathic steatorrhœa shows fatty stools, dilatation of colon, tetany, anæmia, skin lesions and infantilism. The disease occurs in both sexes and history nearly always dates back to early childhood. Serum and plasma phosphorus are low or normal. The total fat in the stools may reach 40 per cent, the bulk of it being unsplit fat.

DIAGNOSIS:—Osteomalacia due to starvation. The case was followed up to 25-4-'52 during which time, the patient was well-fed with a diet of high calcium combined with a sufficiency of vitamin D and exposure to ultra-violet therapy.

Pictures were taken on 25-4-'52 and show the progressive changes that have taken place as a result of treatment.

On 22-4-'52.—Blood calcium was 13.4 mg. Blood phosphorus 8.0 mg.

COMMENT:—It pre-eminently affects women, and is usually rare in men. Rickets and osteomalacia are essentially identical. The difference lies merely in age-incidence; osteomalacia is

adult rickets. Deficient absorption of calcium and phosphorus in infancy leads to rickets. Even in the adult when all bone-growth has ceased, bone is not in a static condition but is constantly being absorbed and reformed. When the absorption of calcium and phosphorus is restricted in the adult a general rarefaction of the skeleton results which is known as "osteomalacia". The essential abnormality is a deficient calcification of osteoid tissue. This deficiency is generalised throughout the skeleton. The cause of osteomalacia was formerly regarded as an endocrine or toxic disturbance. Modern opinion is however, unanimous that osteomalacia is the adult form of rickets in which, as a result of lack of calcium, the lime-stores of the skeleton are depleted in an attempt to preserve the circulation of this essential element.

The deficiency of lime may be due to: (1) defective absorption from inadequate amounts in the dietary; (2) lack of vitamin; and (3) gastro-intestinal diseases, which prevent absorption of lime. It may be due partly to excessive excretion of calcium and it is not unlikely that more than one factor is operative in individual cases. The following clinical varieties are recognised:—

1. *Osteomalacia of pregnancy*:—Though not relevant to the present discussion, there is a relative or an absolute lack of calcium in this condition.

2. *Starvation osteomalacia*:—The cause of this as the name implies, can be traced to starvation and food deprivation and is due partly to an absolute lack of calcium in the diet and partly to avitaminosis.

3. *Osteomalacia of idiopathic steatorrhoea*:—The salient features of this, have been already discussed under differential diagnosis.

One more point about the radiological appearance is that the degree of lack of calcification in the radiographs will vary with the severity of the disease and it is therefore, important to make radiographs, with suitable controls.

All deformities apart from fracture result from weight stress or muscular action.

We are grateful to Dr. Ismail, Superintendent of the Erskine Hospital, Madurai for according permission to publish this case and to Dr. G. A. Naidu, Dermatologist, Erskine Hospital, under whose care the case was admitted for investigation and treatment; our sincere thanks are due to the radiographers and other members of the X-ray staff of the Erskine Hospital for their very active co-operation and assistance.

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6, Vallabhai Road,
Thalakulam, Madurai.

A CASE OF HYDROPHOBIA INITIALLY SIMULATING MALARIA

A. C. ROY, M.B.,

Medical Officer, Charitable Dispensary, P.O. Balagaria, Midnapore.

EARLY on the morning of 19-5-'52 I was called in to see a case with the following complaints:—(1) Fever. (2) Frequent vomiting. (3) Spasmodic backward retraction of the head.

Case history.—The patient a business man used to go to all weekly fairs within a range of 20 or 25 miles to sell or buy goods. He had to sit in the scorching rays of the sun for several hours and in the absence of a conveyance he returned home on foot in the sun. On the afternoon of 17-5-'52 he developed fever with rigor. Prior to that he did not feel well for a couple of days but did his usual work. He vomited several times during the night.

It was reported that on 18-5-'52 the temperature in the morning was normal, from the feel of his body. He was said to have been unconscious for 3 to 4 hours at about noon. Whether he also had then a rise of temperature, his parents could not say.

From morning he was vomiting everything he was given to drink—nothing being retained, even when given in small quantities. From the afternoon there was frequent spasmodic retraction of the head towards the back. Cold water was constantly applied to the head by the drop method.

I saw him early in the morning on 19-5-'52 when I noted the following points:—(1) Age—40, healthy build; (2) Temp. 98.4°F; (3) Pulse-rate—90; (4) Respiration—24; (5) Patient semi-conscious and restless; (6) Frequent vomiting; (7) Tongue—dry and coated; (8) Frequent spasmodic retraction of the head towards the back—the spasms were very frequent coming on every 5 or 10 minutes; (9) Bowels moved the previous day; (10) Passed urine regularly; (11) No other abnormality detected.

A provisional diagnosis of malaria was made and the following treatment was given:—(a) Quinine bi-hydrochlor grs. 10 I.M.; (b) Tr. Ipecac $\frac{1}{4}$ in a tea-spoonful of water at intervals of 15 to 20 minutes; and (c) Gardenal gr. 1—2 doses at an interval of 3 hours.

Cold water to head was to be continued as before. After the injection he vomited only thrice at longer intervals. Backward retraction of the head persisted. In the afternoon the patient complained of difficulty in drinking water, which gradually increased. Thirst was moderate. From mid-night thirst became excessive and though he frequently asked for water he could not drink any. At times he became very restless and required 3 or 4 persons to hold him down.

The parents noticed that every time a glass of water to quench his excessive thirst was shown to him the spasms increased four-fold.

The patient violently resisted the application of cold water to his head. He would not allow fanning to which he did not object earlier. Restlessness continued throughout the night. On 20-5-52, I found him very restless but fully conscious. Temperature—99°F. Pulse—115, Tongue—very dry and coated—beads of perspiration present and increased with increase of restlessness. He insistently asked me to remove the obstacle from his throat so that he could drink water and relieve his excessive thirst but at the sight of water the spasms increased. The symptoms of hydrophobia were now quite evident and enquiry elicited that he was bitten on the left palm by a dog in March last. He did not undergo anti-rabic treatment nor was the wound cauterised. Two other boys were bitten by the same dog at that time one after another—the patient being the first victim. All the three took indigenous treatment for dog-bite. The healing of the wounds was stated to have been uneventful and the dog has been beaten to death.

This was obviously a case of fully developed hydrophobia which initially simulated malaria. The patient died at about noon.

Discussion.—It is very difficult to say whether the case was a case of simple hydrophobia with atypical premonitory symptoms or a case of hydrophobia complicated by malaria. The premonitory symptoms—rigor, fever, vomiting, history of unconsciousness on the 2nd day were all in favour of malaria which is endemic in the area in which the patient lived. Improvement of mental condition and cessation of vomiting after quinine injection would lend support to its being malaria, the spasmodic retraction of the head being a nervous manifestation noticed 24 hours after the onset of fever but not however, usually seen in malaria.

Difficulty in swallowing—a premonitory symptom of hydrophobia was noticed 48 hours after the onset of fever. The earlier symptoms were all in favour of malaria and the later ones in favour of hydrophobia. The provisional diagnosis of malaria was unfortunately not clinched by a blood examination.

Conclusion.—The balance of evidence in this case would therefore, appear to be all in favour of hydrophobia.

Symptomatic relief of Osteo-arthritis and Osteoporosis with Vit. B₁₂

On a weekly dosage of crystalline vitamin B₁₂ of 50 to 100 micrograms, Hallahan treated 33 cases of osteoarthritis with great relief of symptoms. Of 20 of these patients, 7 had obtained 100 per cent relief after one week's treatment. By the end of the 3rd week, 16 showed partial relief of symptoms and 14 had complete relief. By continuing the treatment for longer than three weeks some patients who had obtained partial relief found 100 per cent relief. No toxic side effects were noticed in any case.—(*Amer. Pract. and Dig. Treat.*, 3:1, 27-32, 1952).

A CASE OF 'SCRUB TYPHUS' AT SAGURNAL TEA ESTATE

K. N. DAS, L.M.P., D.T.M.,

Assistant Medical Officer, Sagurnal T.E., Sylhet Dist., East Pakistan.

K., a Hindu girl, aged 14 years, was on the rolls of the 'out-patient' department for a remittent type of fever since 18-5-'52. Her blood for M.P. was negative and antimalarial therapy had no effect. She was then admitted into the estate hospital on 24-5-'52.

Brief clinical features of the case

The onset was sudden with a moderate degree of fever, headache and pain all over the body. The fever was more or less remittent all through and terminated by rapid lysis after aureomycin therapy. The headache was frontal and severe during the first week of illness. Later on it subsided. The mental condition was dull with a drowsy look. Though she was fully conscious, she could not answer questions readily. This slight degree of deafness completely disappeared when the temperature came down to normal; a mild low muttering type of delirium was noticed for only a day during sulpha and penicillin course. Insomnia and tremors of lips, fingers and forearms were present only towards the end of the second week of fever. There was no skin rash or eschar, nor any lymphadenitis. The spleen was palpably enlarged, while the liver was not palpable. A mild bronchial catarrh was noticed during the second week. The pulse varied from 110 to 138 p.m. V/T was thin/low. The heart had a hæmic murmur +++. (Patient was a long standing case of anæmia with 30% Hb. "Tallqvist Scale").

The tongue was slightly coated, and constipation was marked during the first half of illness. Later on the bowels were somewhat loose. There was no nausea or vomiting; a trace of albumen was present and the *diazo-reaction for typhoid* was negative.

Other Laboratory Findings—

BLOOD COUNT

Case No.	Total count	B.	E.	M.	J.	St.	S.	L.	Large mono	Remarks
—	—	—	—	—	3	21	31½	39	5½	—
						55½				
M. Parasite not seen		Blood Picture								

Polychromatophillin + + + ; Poikilocytosis + + ; Anisocytosis + + ; Normoblasts + + ; and Megaloblasts + .

Widal tests on 26-5-'52, 2-6-'52, and 20-6-'52 were all negative. Weil-Felix reaction: Ox 19 and Ox 2—negative ; but Ox K—positive, Titre 1/250.

but it went up to 101°F, next morning. Aureomycin was continued as before for 4 days and the temperature came down to normal by rapid lysis (*vide temperature chart, p. 208*). It was then continued 250 mg. t.d.s. for another 2 days and then discontinued. After the aureomycin course, she was put on a vitamin B-complex tonic and her convalescence was rapid and uneventful.

Points of interest.—1. Typhus fever is not common in this district. This case was reported to the District Civil Surgeon and an enquiry was made regarding any previous incidence of typhus in this district. The Civil Surgeon replied "No case of typhus fever has yet come to our knowledge". 2. It may be of interest to tea gardens in this valley because of its rarity. It also gives a warning that neglect of the routine Weil-Felix test in all typhoid-like fever cases may land one in error of diagnosis. 3. The source of infection could not be traced out definitely. [In the same lines, where this patient lived, a woman died on 20-5-'52, on the twelfth day of fever from intestinal hæmorrhage. She had well marked deafness, mental dullness and apathy with persistent headache. Her blood showed no M.P.; Widal report received after her death was negative. W.F. test could not be done due to shortage of reagents. Could it have been typhus? if so, is not intestinal hæmorrhage an uncommon feature in typhus?]. 4. It is also interesting to note the striking therapeutic value of 'aureomycin' as in a rickettsial disease.

Acknowledgement.—My thanks are due to Dr. C. H. Phillips, M.O. of the Estate for valuable help, in investigating the case, to Mr. J. Turney, Manager of the Estate, for kind co-operation and permission to publish the report, also to Dr. Q. A. Khaleque, of the Dacca Medical College and Dr. A. C. Chakravarty, of the Juri Valley Central Laboratory, for helping me with the laboratory examinations.

Reference :

Indian Medical Gazette, Vol. lxxxiv, No. 2.

Pneumothorax in Newborn Infants

Pneumothorax may cause the death of a newborn infant without a diagnosis. This may occur in premature as well as in full-term infants. Trauma, violent artificial respiration and oxygen insufflation without controlling oxygen pressure are usually the causal factors. Dyspnoea and cyanosis are characteristic symptoms. Shock may follow rapidly. The diagnosis should be suspected in the presence of these symptoms associated with rales in the base and middle zone of the lung. The diagnosis is confirmed immediately by radioscapy and X-ray examination of the chest (front and profile views). Treatment of this emergency consists of immediate puncture of the thorax with slow withdrawal of air from the pleural space. Correa and Barrani of Montevideo report on a case which responded favourably to this treatment.—(*Hoja Tisiol*, 11, 239 *Abst. in Jour. Am. Med. Assoc.*, 9-2-1952).

A TYPICAL CASE OF TOXIC GOITRE

V. R. KULKARNI, L.C.P.S. (Bom.),
Regd. Medical Practitioner, Chalisgaon (E.K.)

A PATIENT came to me with the following complaints: (1) Cough; (2) dyspnœa; (3) fullness in the stomach—epigastric region—more marked on taking even liquids; (4) pain and tenderness over the hepatic region; (5) scanty urine; and (6) diarrhœa.

Patient's general history.—A Marvadi by caste of sober habits, but given to excessive smoking and tea drinking; irritable and nervous by nature; pale and thin in appearance and build strictly vegetarian.

Physical examination:—Tremors of hands and feet. *General*:—Anæmia, exophthalmos, enlarged thyroid, swelling on face and feet. Heart and pulse:—Tachycardia 80 p.m., heart dilated, apex beat in 7th intercostal space and on outer side of the nipple line. Lungs:—Rales and rhonchi all over the lungs. Liver:—Painful, tender and palpable, along the costal margin. Abdomen:—Soft but tender with colicky pains and diarrhœa—frothy stools. Urine:—Scanty 10 to 12 oz. per day; high coloured. Sugar absent. Albumin + + +. Blood pressure:—Normal. Taking into consideration: Tremors, enlarged thyroid and exophthalmos, Von Grave's sign was tested and found positive.

Provisional diagnosis:—Secondary dilatation of the heart resulting in back pressure in the lungs, liver and kidneys, on account of primary toxic goitre.

TREATMENT:—Though the case was one of toxic goitre the condition of the heart and kidneys was treated first to avoid serious complications or permanent damage.

(1) Aminophyllin 10 c.c. I. V. daily for 6 days; (2) Procaine penicillin G 4 lac units, daily for 3 days; (3) diuretic mixture—

℞	Pot. citras	... 3 i
	Diuretin	... 3 p
	Tr. Digitalis	... 3 p
	Tr. Scillæ	... 3 p
	Ext. Funarnava liq.	... 3 i
	Ext. Glycyrrhiza liq.	... 3 i
	Aqua ad	... 3 iii

(4) Daily dose of Mag sulph early morning for drainage if bowels did not move; (5) Minaphil tablet 1 t.d.s.; (6) Calci Diuretin (Knoll) 1 tab. b.d.; (7) salt free diet; and (8) complete rest in bed.

With this line of treatment the patient improved very well. (1) Oedema on the face and feet disappeared; (2) quantity of urine increased to 40 oz per day. Albumin also disappeared; (3) he could retain fluids and then gradually solids also; and (4) dyspnœa cough, fullness in the epigastrium subsided.

Later on full diet was resumed with chloride-free table salt.

Though complete rest in bed was enforced, the patient on feeling better started moving about; consequently got a fresh attack with (1) acute diarrhoea and vomiting; (2) severe tachycardia—pulse rate 150 p.m., other systems however, being normal.

TREATMENT:—(1) Ice pieces to suck, plain boiled water by mouth; (2) I. V. Glucose 25 c.c. and saline (normal) 200 c.c.; (3) Aureomycin 2 capsules every 6 hours; (4) Formo-Cibazol 2 tab. every 4 hours; (5) Digitanid (Sandoz) π xv t.d.s. until the pulse rate was 80 p.m. Later on the regular treatment for toxic goitre was started:—(1) Methyl thiouracil—tablets (just before meals) 1 gm. tab. t.d.s. for 2 months; (2) Lugol's Iodine—4 drops in an ounce of milk, before meals for 4 months; (3) Calcibronat (Sandoz) granules 2 teaspoons in a cup of water b.d. half an hour before meals for 2 months; (4) Dayalets (Abbotts) tab. and Beplex (A.F.D.) tab. one each morning and evening with milk for 2 months; (5) Absolute rest in bed for 2 months; (6) Injection I.M. Liver extract 1 c.c. (Lederle 15 U.S.P.), Berin 1 c.c. (100 mg.), Pyridoxin 1 c.c. (Endo) mixed together daily for 10 days and then on alternate days

(Continued on page 212)

Obesity—A Simple and Effective Treatment

Dr. David Halperin of Westminster Hospital, London, who has made a special study of this subject found clear evidence that obesity is associated with retention of water, which is partly caused by retention of chloride. A great deal of evidence suggests that white wheat flour is a factor in maintaining this water retention. Incidentally it is a fact that carbohydrate such as rye or barley or rice has no such anti-diuretic effects; it is therefore, suggested that the bulk in the diet be made up by material containing one or other of these products.

The loss of weight can be assisted in four ways:—(1) By reducing the actual intake of fluids to 2 pints per day. (2) By forbidding the use of added salts. (3) By forbidding the use of wheat-flour products. (4) By the administration of an oral diuretic in the form of urea.

If the weight loss fails to be maintained at 1 lb per week then the patient's morale can be assisted by a small intramuscular injection of a mercurial diuretic. There is a great deal of evidence to show that excessive weight is associated with a diminished expectation of life. Loss of weight can be achieved without severe dietetic restrictions by the curtailing of fluid intake, by the curtailing of sodium chloride intake and avoidance of wheat flour products. This will produce a loss of weight of not less than 1 lb and not more than 2 lbs a week. Such a weight reduction is more likely to prove of lasting benefit to the patient than one which has been suddenly and dramatically inflicted on him by the more drastic and irksome types of diet ordinarily prescribed for the purpose.—(*Med. World.*, 19-12-1952).

for 10 injections. Total 20 injections; and (7) Fried and greasy food to be avoided.

Dr. Kohiyar, M.D., of Bombay advised that deep X-ray exposures should be given in addition to the above treatment, but it was postponed until the treatment given above was carried out, as the radiologist was of opinion that deep X-ray therapy should be given only when surgical treatment is not contemplated. Otherwise, surgical treatment, found necessary a later date, will become very difficult, owing to the deep X-ray exposures given previously.

RESULTS :—The patient improved most uneventfully afterwards under the above treatment and gained in weight, with good appetite and digestion. Tachycardia completely disappeared. Von Grafe's sign, tremors and exophthalmos absent. Excepting the apparent enlargement of thyroid the patient has no complaint whatsoever nor any signs and symptoms of toxic goitre. The patient is now leading a normal and cheerful life. I am grateful to Dr. Kohiyar of Bombay and Dr. Kirtane of Dhulia for giving valuable advice whenever sought for.

*'Raghunath Nivas',
Dhulia Road,
Chalisgaon [E.K.].*

The Sedimentation Rate in Myocardial Infarction

The sedimentation rate in coronary occlusion and myocardial infarction is a most useful diagnostic procedure. Dr. Milton Plotz of the State University of the New York School of Medicine carried out an investigation to confirm the accuracy of this method, and to evaluate its usefulness as a guide in managing patients, especially with regard to such matters as getting the patients out of bed and exercise. One hundred consecutive cases of major myocardial infarction as confirmed by E.C.G. changes or *p m.* examination are included in this study. The Wintrobe method was used in most cases, with correction by the haematocrit determination.

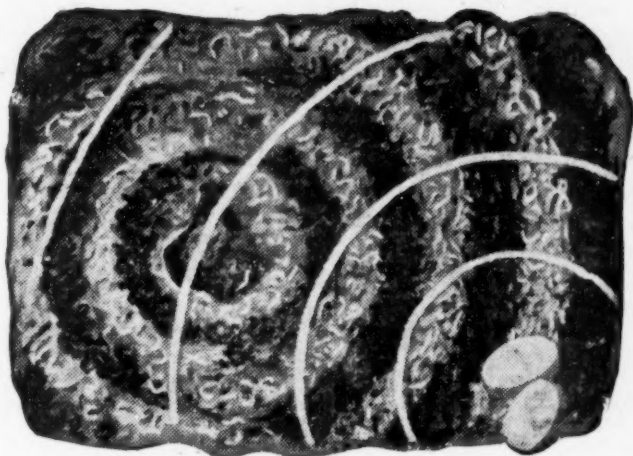
(1) ESR was elevated in 97 of the 100 proved cases of major myocardial infarction.

(2) ESR is a highly useful diagnostic procedure especially when the temperature is elevated little, if at all.

(3) The rate is seldom elevated before 48 hours after infarction but is increased in almost all cases after 72 hours; the elevation persists for 4 to 8 days and in most cases the rate is back to normal by the 36th day.

(4) In 76 of 97 cases the rate became normal in 36 days, in the other twentyone, 3 had first infarction, 13 had disease outside the heart, and in 5 there was evident explanation for persistence of E.S.R.

(5) In a few cases the rate is persistently elevated after patient has become clinically well; in such cases he may be allowed out of bed cautiously on the 42nd day.—(*A. J. Med. Sci.*, July 1952, pp. 23-26).



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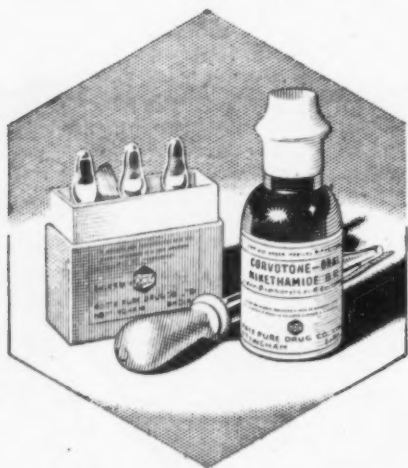
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No. 3

DRIVE AGAINST TUBERCULOSIS

CENTRAL AND MADRAS STATE MINISTERS' APPEALS

PRESIDING over the B.C.G. Day celebrations at the Madras Medical College on the 23rd February '53 Shri Dr. U. KRISHNA RAU, Minister for Industries, Labour and Transport, Madras State, appealed to the public to co-operate with the Government in its efforts to fight against Tuberculosis by popularising B.C.G. vaccination, which was a harmless and effective preventive measure. The Minister for Health, The Director of Health Services, the Director of the B.C.G. Vaccine Laboratory and the Director of the Tuberculosis Institute of Madras were also present and explained the technique of manufacture and the details relating to the vaccination and its effectiveness in preventing Tuberculosis. "The main causes for the spread of Tuberculosis" said Dr. U. KRISHNA RAU "were the poor economic conditions, lack of proper housing facilities and malnutrition due to inadequate food. The Madras State Government had undertaken B.C.G. vaccination with the help of the Union Government; the WHO and UNICEF had also contributed liberally in men and money to the State Government's attempt. In Norway T.B. was completely rooted out by B.C.G. vaccination. In the Madras City children studying in more than 135 schools maintained by the Corporation have been B.C.G. vaccinated, as also many employees in firms and institutions. B.C.G. has now received universal acclamation and is considered by prominent medical men all over India as an effective measure for prevention of tuberculosis".

Mass B.C.G. vaccination can be a success only if the whole-hearted and enthusiastic co-operation of the public and of the

medical men was spontaneously forthcoming. Legislative enactments and their enforcement will not succeed half as well as active and willing co-operation from the two sources mentioned.

Dr. KAR EVANG, Vice-Chairman of the visiting team of 14 leading medical scientists who stayed in Madras city for a month from the 6th February '53, who is also the Director-General of Health Services in Norway stated that when B.C.G. vaccination was first introduced, there was opposition from some of the conservatives amongst the medical men in Norway. But in about 20 years B.C.G. vaccination became very popular and everybody began to ask that his child should be vaccinated. That was the turning point. After the Second World War, B.C.G. vaccination was started in Europe in 1947 after carefully planned trials and millions of people in several European countries took advantage of this procedure. The Conference of Medical Experts who met in Delhi in April '51, recommended a campaign of B.C.G. vaccination in India. The Union Health Minister, Srimathi RAJKUMARI AMRIT KAUR broadcasting from Delhi on the eve of the B.C.G. day celebrations last month, also pleaded strongly for public co-operation with the Government in implementing the B.C.G. vaccination campaign. Up to the end of 1952 over forty lakhs of persons had been vaccinated in India. The Union Health Minister said "Tuberculosis has been assuming epidemic proportions and has been estimated to kill a person every minute in India. Next to Malaria this is the biggest killer to-day and 5 lakhs of people are dying of tuberculosis every year and atleast five times that number are suffering from the disease all the time". This is certainly a very serious matter in all conscience and every well-tried and successful method of protection against the disease should therefore, be welcomed and readily adopted. The unwarranted criticisms and opposition launched in certain quarters against B.C.G. vaccination, by persons who readily acquiesced in the sophistry and casuistry of a few prejudiced, if well-meaning members of the medical profession both here and elsewhere, is luckily fast dying out, and people have already taken to it kindly, as evidenced by the numerous B.C.G. vaccinations already carried out in many States of India.

The mass campaign was started in April 1951 in the U.P., Punjab, and Madhya Bharat. The B.C.G. vaccine and the tuberculin solution used in this mass campaign are manufactured at the B.C.G. Vaccine Laboratory at Guindy (Madras) by experts who have received specialised training at the State Serum Institute in Copenhagen. This laboratory went into production in Feb. 1949 and is today ranked as one of the biggest BCG production-centres in the world. A million doses of vaccine and one and a half million doses of tuberculin solution are manufactured every month at this Institute. "The programme for 1953 envisaged" said the Union Health Minister replying to Srimathi Renu Chakravarthi in the House of the People

at Delhi on the 23rd Feb. 1953, "that mass campaign would be started in Delhi, Hyderabad, Bombay, Jammu, and Kashmir, Madhya-Pradesh, Madras, Orissa, Saurashtra, Rajasthan, Manipur, Coorg and Tripura." It has been estimated that there are in India about 17 crores of persons below 20 years of age, the majority of whom need B.C.G. vaccination. If tuberculosis is to be eradicated, or at all events brought under effective control, there should be a powerful and organised drive on the part of the Health authorities in all the States of India to secure willing and hearty response from the people. Any opposition from interested parties must be effectively countered by suitable propaganda.

May we not hope that, notwithstanding the serious handicaps and obstacles existing in our country and the apathy and ignorance of the majority of our brethren in matters of health, it would still be possible to do a considerable amount of spade work in the next few years and ultimately speed up the progress of B.C.G. vaccination so as to achieve signal success in eradicating tuberculosis from our midst?

SEMINAR ON MEDICAL EDUCATION:

CONFERENCE OF WORLD MEDICAL SCIENTISTS

AT the two-day seminar on Medical Education, which opened at the University Buildings, on the 2nd March 1953 *in camera*, discussion was held with particular reference to under-graduate education in basic medical science.

Members of the WHO team, representatives of the Medical Faculties of 15 Indian Universities, Members of the All-India Medical Council, and of the Central Medical Institute, representatives of the Rockefeller Foundation and the Deans and Professors of Medical Colleges attended.

Dr. GRZEGORZEWSKI, Director of the WHO Division of Education and Training Services, gave an account of the trends in medical education and the steps being taken in different countries to study problems connected with medical education. The Conference appears to have laid emphasis on the need for general education in Physics, Chemistry and Natural Science, in the pre-clinical stages and stressed the importance of anatomy, physiology and biochemistry.

Another aspect emphasised related to preventive medicine. Preventive and social medicine formed an integral part of study. Opportunities for training in field-work during the under-graduate course was important, so that the general practitioner could have some definite idea of his responsibility in the field of preventive medicine.

Discussing the importance of diagnosis as compared with treatment, it was stated that with modern discoveries in therapy, the need was to lay stress on the essential methods of diagnostic procedures; the newer methods of treatment could then be followed more easily, once the fundamentals of diagnosis were correctly appreciated.

Most colleges in the USA employed full-time salaried doctors. In some cases, they were given an opportunity to attend on cases in hospitals, on a limited scale. But that was likely to be abused, and so it was tolerated to the extent that government was unable to pay adequate salary to full-time teachers. Teaching could be done better if adequate salaries were given to full-time teachers, but it was not desirable to have all of them as full-time teachers as some part-time teachers, who had consulting practice, had a definitely valuable contribution to make as clinical teachers. In some countries the whole staff was full-time, except perhaps, the Head of the Department, who might be allowed some consulting practice.

In the Nationalised Medical Services in the United Kingdom, the salaries of teachers of the basic and pre-clinical sides had been upgraded to the level of clinical specialists. The best plan would be to have full-time staff of some proportion, and a fair amount of part-time staff to give the benefit of their experience in their respective fields to the students.

Postgraduate medical education was discussed on the second day of the Seminar. The training of house physicians, refresher and short-term courses, and the policy of continuing education for the staff of hospitals, formed the main items. Dr. John Gordon of Harvard initiating the discussion, said medicine was a continually expanding science, and the medical profession had therefore, to follow a life of perpetual studentship.

Dr. OSMOND CLARKE of the London University described the system in vogue in England. It was compulsory for everyone to undergo a house surgery course for a year. Some stayed on to take up a junior registrar's post for a year on a salary (about Rs. 5,300 to Rs. 6,700). They then left for general practice, or took up a medical registrarship for a year on a salary of about Rs. 12,000. Most of them would get a higher qualification for membership or fellowship, and then go out for general practice. Many other hospitals, besides the teaching hospitals, were recognised for the training of house physicians and house surgeons, and the extent to which they were utilised depended on the facilities afforded. A good deal of the training was given by the resident staff. The obligation on the part of the hospital was to train the house physician not merely to use him for the work of the hospital but as a useful person for the work of the department concerned.

It was suggested that at least two months' time of the house physicians and surgeons should be devoted to public health training in a rural set-up; when they would be trained by a public health officer of recognised capacity. The house physician should *not* be utilised as another addition to the staff for routine work.

The next point emphasised was the importance of training in clinical investigation, and the development of capacity for research among those who would be resident for some years, and were therefore, better prepared to assume this responsibility. The starting of refresher and short-term instruction in smaller communities and rural areas was discussed. The difficulty in teaching practising doctors was that those who most needed it, might not and usually did not attend these courses. Good teachers, who could find time, were not easily available. Refresher courses, lasting a week in the capital of the State once or twice a year, were being held.

In the course of the discussion that followed it was brought out that such refresher courses had been arranged by the Post-graduate Councils of Madras and Andhra Universities for the past seven years. It was stated by the Director of Medical Services, Madras that a scheme had been implemented by his department to have these refresher courses in the districts, groups of districts being chosen every year in rotation. It was also stated that no refresher course had been arranged for public health officers in India, and such courses should be arranged for public health officers.

The Conference then considered the subjects of advanced training in medical science, and in the clinical specialities. It was suggested that some kind of selection might be attempted even during the under-graduate stage, and the person systematically helped to develop those faculties. Certain departments might be upgraded, and declared to be suitable centres for post-graduate study.

Even if there were excellent training institutes, it was good to send practitioners abroad to study in foreign universities provided the young doctor had training previously in his own centre.

In the discussion on training in clinical specialities, it was stated that the training might extend to four or five years depending on the speciality concerned.

The system of granting fellowships and travel grants by WHO was explained by Dr. GRZEGORZEWSKI, Director of Medical Education in WHO. Winding up the discussion, he stressed that the subject of medical education was one of international study, and such discussion would promote the healthy growth of ideas.

Eminent medical men from all over India participated in the seminar.

We have been consistently advocating in these columns, for several years, reforms in medical education on the lines discussed at the Seminar and we are therefore, in full accord with the decisions

arrived at. On the question of foreign travel for advanced studies however, we feel that after the upgrading of our medical colleges in India, post-graduate medical education should be confined to this country. Foreign travel may, however, be undertaken by medical men on their own, when they have settled down in practice, if they wish to widen their sphere of professional knowledge by observing the procedures and techniques in use in other lands.

VISITING TEAM OF MEDICAL SCIENTISTS

Fourteen of the world's leading medical scientists were in Madras City from the 6th February and the tour was sponsored jointly by the World Health Organisation and the Unitarian Service Committee Inc. U.S.A. The purpose of the visit was to help Indian Scientists and to take part in discussions on the various aspects of medical science. The team was composed of the following :

Dr. Einar Lundsgaard, Professor of Physiology, University of Copenhagen, Denmark.

Dr. Corneille Heymans, Professor of Pharmacology, University of Ghent, Belgium.

Sir Alexander Fleming; Principal, The Wright-Fleming, Institute of Microbiology, University of London, Great Britain.

Dr. Joseph C. Aub, Professor of Research Medicine, Harvard University, Boston, Mass., U.S.A.

Dr. Samuel Z. Levine (*Chairman*), Professor of Pædiatrics, Cornell University, New York, N.Y., U.S.A.

Dr. Pietro Valdoni, Professor of Surgery, University of Rome, Italy.

Dr. Erik Husfeldt, Professor of Surgery (Thoracic Surgery), University of Copenhagen, Denmark.

Dr. Henry Osmond-Clarke, Honorary Surgeon and Assistant Director, The Orthopaedic and Accident Hospital, University of London, Great Britain.

Dr. Georges Portmann, Professor of Oto-Rhino Laryngology, University of Bordeaux, France.

Dr. Stuart C. Cullen, Professor of Anæsthesiology, The State University of Iowa, Iowa City, Iowa, U.S.A.

Dr. Leo G. Rigler, Professor of Radiology, University of Minnesota, Minneapolis, Minnesota, U.S.A.

Dr. Edward Grzegorzewski, Professor of Public Health, Director of W.H.O. Division of Education and Training Services, Geneva, Switzerland.

Dr. Karl Evang (*Vice-Chairman*), Director General of Public Health of Norway, Oslo, Norway.

Dr. John E. Gordon, Professor of Preventive Medicine and Epidemiology, Harvard University School of Public Health, Boston, Mass., U.S.A.

The team was led by Dr. Samuel Z. Levine, with Dr. Karl Evang as Vice Chairman. They were in the City from the 6th February and left for Bombay on the 5th March. During their 4 weeks' stay in Madras City they gave interesting lectures and demonstrations to the members of the medical profession and took part in discussions on various subjects. These lectures were greatly appreciated by all who attended and the film shows and slide demonstrations that one witnessed were really a treat. We trust that such visits by eminent medical men from foreign countries will henceforth become a common feature so that Indian doctors may have an opportunity of comparing notes and exchanging views for mutual benefit.

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MEDICINE AND THERAPEUTICS

The iron losses of healthy women during consecutive menstrual cycles.—(*Am. Jour. Digest. Dis.*, 19: 6, June 1952 from *Med J. Austral.*, 1951, p. 669).

The iron losses were determined during consecutive menstrual periods in a group of 14 normal healthy young women and pronounced individual variation was observed. The average loss was 21.8 mg. of iron (range being 3.2 to 66.8 mg). The iron losses in the same subject varied from one menses to another. The average duration of the cycle was 28.7 days (range being 21 to 55 days). A number of women are situated very unfavourably for the maintenance of iron equilibrium and in these women, it seems very likely that anaemia will gradually develop.

Erythromycin —(Leading article, *Br. Med. Jour.*, 15.11.1952).

Erythromycin (also named Ilotycin) is obtained from a strain of *Streptomyces erythreus* found in a soil sample from an island in the Philippines. Mc Guire and his co-workers announce its discovery and describe its chemical and physical properties and its antibiotic activity. Heilman *et al* (*Proc. Mayo Clin.*, 27, 285, 1952) give a general account of the behaviour of this new drug *in vitro* and of the effects of its use in various patients in the Mayo Clinic. Haight and Finland (*New Eng Med. Jour.*, 247, 227, 1952) describe a similar study in Boston City Hospital.

Erythromycin is a basic substance soluble in water to the extent of 2 mg. per c.c. reasonably stable and much more active in an alkaline than an acid medium. It has a bacteriocidal as well as bacteriostatic action: bacteria can acquire resistance to it apparently with some rapidity—but this resistance is unrelated to the resistance to other antibiotics. Its spectrum resembles that of penicillin, gram-positives and a few of

the more fastidious gram-negatives (*Neisseria* and *Haemophilus*) being sensitive and coliforms resistant. As with penicillin, haemolytic streptococci are highly sensitive and staphylococci rather less so: on the other hand, corynebacteria are exceptionally sensitive to erythromycin, and it is of some significance that Haight and Finland have cleared three persistent diphtheria carriers with it. The drug is administered by the mouth and behaves pharmacologically like the other new antibiotics: therapeutic concentrations can easily be maintained in the blood but at the same time, whether owing to incomplete absorption or to biliary excretion, there is a pronounced effect on the faecal flora, gram-positive elements including clostridia being suppressed and coliforms unaffected. Daily dosage has varied from 1 to 3 g. and dosage intervals from 3 to 6 hours. The only factor limiting dosage is a tendency of the drug to cause vomiting and diarrhoea, no other toxic effect having been seen. Therapeutic results in pneumonia and in streptococcal throat infections have been as good as those obtainable with other antibiotics. A more important use for erythromycin is suggested by the successful treatment of two cases of staphylococcal septicæmia (one in each series) in which the organism was resistant to antibiotics. On the other hand, failure in four out of five cases of endocarditis suggests that the capacity of bacteria to acquire resistance to this drug may intervene here: such a change was actually demonstrated in two of these cases.

First impressions are that erythromycin will be a valuable agent to hold in reserve for infections resistant to everything else. The general tendency among staphylococci and coliform bacilli to become resistant to the newer antibiotics as well as the old is illustrated in the findings of Thompson (*Med. J. Aust.*, 1952, 1, 870) who mentions incidentally that three patients at the Royal Prince

Alfred Hospital, Sydney—two with pneumonia and one with a totally resistant staphylococcus—all died. This gradual process of bacterial habituation to drugs during the years following their discovery makes it all the more necessary for fresh ones to be forthcoming at intervals, and it is reassuring to know that we have not reached the end of Nature's resources in this direction.

A leading article in the '*Antibiotic and Chemotherapy*', 2: 279, 1952, containing the first announcement of this discovery mentions some interesting facts. It is well known that several firms in the U.S.A. are conducting extensive soil-screening programmes on samples from all over the world with a view to discovering new antibiotic-forming species. Erythromycin is of course, the product of such a survey and Eli Lilly & Co. are now added to the list of those fortunate concerns who have discovered a winner. But it is here stated that the number of firms engaged in such screening programmes is 16. Although the extent of these operations and the skill with which they are conducted are important, chance is perhaps still more so; a lucky dip on such a scale has never been seen before in anything connected with medical science. It is also mentioned that the monthly American production of penicillin is now 30 tons, of streptomycin 21, of aureomycin, chloramphenicol, and terramycin 24, while that of sulphonamides is surprisingly enough, on the up-grade also. It would be interesting to know what proportions of these vast quantities of antibiotic (enough to supply a 10 g. course to over 90,000,000 patients annually) are being used for necessary purposes.

Sciatica.—(*Rev. Assoc. Med. Argentina*, Eng. Abst. *J.A.M.A.*, 2:2-'52, p. 411).

Genito-urinary disorders, congenital spinal anomalies, flat feet, bony alterations in feet and legs, osteoarthritis of the lumbar spine, radiculitis of various kinds, infectious diseases like typhoid may give rise to low back pain. The pain simulates that caused by disc pressure whether there is radiation along the trunk of the great sciatica or not. Costa

Bertani believes that a thorough examination of the patient will reveal the true cause of the pain. *This examination must be made by a general practitioner and not by a specialist*, because the G.P. is in a better position to evaluate the data obtained. Careful interrogation and a complete history will help in making a correct diagnosis. The treatment of choice is medical, surgical and orthopaedic treatments have special indications. Eighty per cent of Bertani's patients were relieved by conservative medical means. Correction of an underlying cause will often cure even long standing sciatica.

Clinical trials in India with tibione in pulmonary tuberculosis.—(Nagpaul, *D.R., Ind. Jour. Med. Res.*, 40: 2, April 1952).

In December 1950, the Director General of Health Services Govt. of India supplied 500 gms of Tibione (P. Acetyl-amino-benzaldehyde-thiosemi-carbazone) in tablets of 50 mg. each for trials in patients with pulmonary tuberculosis in the Lady Linlithgow Sanatorium, Kasauli. Dr. Nagpaul conducted a well planned and controlled experiment more or less on the lines of the Br. Med. Res. Council's trials on streptomycin. 41 patients were selected by a team of 4 doctors, and all the patients had predominantly exudative disease, with pyrexia of 100° F. or more, malaise, loss of appetite and sleep, a raised ESR (10 mm over normal-Wintrobe) and sputum showing a.f.b. The selected patients were allocated either to the "trial" or the "control" group at random, by drawing lots.

The daily dosage in 3 equally divided doses was given after meals, adults received an initial dose of 50 mg. a day for 3 days, and if they tolerated it 75 mg. for another 3 days and raised to 150 mg. daily. If not tolerated the dose was slowly built up starting with 5 mg. a day. For children below 15 half the adult dose was used. Total period was 120 days in each case unless toxic symptoms demanded a discontinuance of the treatment. The controls (20 patients) received vitamin C tablets only for psychological reasons. The youngest patient was 12 years old and the oldest

was 50: Between 20 and 35 years there were 14 in the trial group and 12 in the control group.

Tibione appeared to have definite toxicity, greater than PAS and streptomycin. Toxic symptoms disappeared with reduction in dosage or discontinuance of the drug. Anorexia was the most frequent and persistent troublesome side-effect, and invariably it came up when the optimal dose was reached; severe constipation was noticed at varying intervals. Albuminuria occurred frequently in our cases at intervals. In two diabetics the albuminuria persisted, while in one other diabetic there was no albuminuria at all.

No significant beneficial effects of the drug was noticed in this experiment, and it proved to be a potentially toxic drug.

Treatment of sprue with folinic acid.—(*Am. Jour. Med. Sci.*, 224: 1, July 1952).

Folinic acid is an active principle isolated from enzymatic digests of liver in 1919 by Bond, Bardos, Sibley and Shive (*J. Am. Chem. Soc.*, 71: 11), who showed that folinic acid was 10 to 100 times more active than folic acid in overcoming the inhibition of certain bacteria, caused by the folic acid antagonists. This active principle is identical with a factor present in varying concentrations in commercial liver extracts which was shown to be necessary for the growth of the organism *L. citrovorum*, by Sabberlich and Baumann in 1948 (*J. Biol. Chem.*, 176: 165). The first clinical report of the use of folinic acid or the citrovorum factor was by Spies and his coworkers in December 1950 (*South. Med. Jour.* 43: 12, 1950). Subsequent reports by Davidson and Girdwood, Ellison *et al*, and Meyer *et al*, were on its successful use in pernicious anaemia, and Woodruff and his coworkers reported on its usefulness in megaloblastic anaemia of infancy.

Quite recently Suarez, Martinez and Suarez of Puerto Rico obtained very good results in 2 cases of sprue. In view of the paucity of more adequate trials and reports on the use of the citrovorum factor or folinic acid in

sprue, Romero, Vizcarrondo, and Rodriguez-Molina, all of San Juan, Puerto Rico, selected a group of nine patients and closely obtained their clinical and hematological response to intramuscular folinic acid. All the nine were confirmed by all known clinical and laboratory findings to be genuine cases of sprue. None received any hematronics for at least 10 weeks prior to the starting of folinic acid therapy. On admission they were given a basic diet, with no meat or eggs which corresponded to the average diet of low income groups in the state.

(1) Three cases received 100 to 400 units of folinic acid (supplied for this clinical study by Dr. W. C. Young of Eli Lilly & Co in the strength of one unit being equal to one microgramme of pure citrovorum factor) intramuscularly for 12 days. All the three showed clinical and hematological improvement which was however, considered inadequate by the authors.

(2) Six other cases of sprue received 1000 daily units of folinic acid by intramuscular injection for 12 days: (a) the clinical symptoms of the disease were rapidly and effectively controlled in all cases (b) the hematological response was quite adequate in five and sub-maximal in one.

(3) There were no untoward effects from the administration of folinic acid by the deep intramuscular route.

(4) Folinic acid is qualitatively as effective as folic acid in the treatment of sprue. Weight by weight this substance appears to be 10 to 15 times more active than folic acid in the treatment of sprue and may well represent a derivative of the latter agent which retains all of its anti-sprue activity.

The climacteric and oestrogen therapy.—(Editorial, *Br. Med. Journal*, 3rd Jan. 1953, p. 37).

The primary cause of the menopausal syndrome, as it is sometimes called, is ovarian failure, but most of the symptoms are not due directly to this but rather to a secondary disturbance, operating, through the pituitary gland

of the whole endocrine balance and of the closely linked autonomic system. The ovarian failure can be countered by oestrogen therapy and indeed each year brings new commercial preparations of synthetic and natural oestrogens which are advocated for the relief of menopausal symptoms. Most recently the superior merits of combining oestrogen with androgen for this purpose have been widely publicised. The two hormones are in many ways complimentary and synergistic in their actions, but they do not neutralize each other, and when given together undesirable side-effects of both may appear simultaneously—vaginal bleeding alongside hirsutism and hoarseness. Unfortunately the claims made for some of the new hormone preparations have been more enthusiastic than discreet, and the range of symptoms often in fact coincidental, which are claimed as indications, is being increasingly widened. In addition, with little or no change of proprietary name, oestrogen has been added to the formula of preparations previously free of hormones. And, as oestrogen is readily absorbed through the skin, the use of skin creams containing only minute amounts may cause undesirable side-effects. One practical consequence of all this is that oestrogen medication, sometimes without the patient or the doctor being aware of the exact constitution of the prescribed medicine, is becoming one of the commonest causes of post-menopausal bleeding. It suggests that the temptation to resort to oestrogen as a first line of treatment is becoming increasingly difficult to resist.

In only about 20% of women are

menopausal symptoms severe. In the majority of cases, simple explanation of cause and significance, with due emphasis on the temporary nature of the upset, and repeated reassurance by the family physician to counter the common feeling of insecurity are sufficient. In many cases the judicious prescription of mild, sedatives also helps. In a minority, probably less than 10% in whom severe discomfort persists despite these measures, oestrogen therapy is indicated with the object of tiding the patient over the period of adjustment. If this is to be achieved without side-effects troublesome to the patient and without irregular bleeding worrying to the doctor, certain principles should be observed:—

Strict instructions about dosage are necessary, otherwise the patient may resort to an occasional tablet when unwell or unhappy and so develop an oestrogen resistance. *The oestrogen of choice should be given orally and in the smallest dose which just fails completely to relieve all symptoms.* It should be administered for 21 consecutive days, out of 28, thus simulating the normal cycle, and treatment should not be discontinued suddenly but gradually tapered off over 3 to 4 months. Any abnormal bleeding which accompanies or follows treatment must not be regarded as consequential, but the patient should be referred for full gynaecological examination including curettage. Indeed, where there is a strong family history of genital cancer or where the menopause was induced by surgery or radiotherapy for gynaecological carcinoma or endometriosis, *oestrogens are probably best avoided.*

SURGERY

Lymphoedema of the lower extremities—(*Virg. Med. Monthly*, 79:7, 351-362, 1952).

Lowenberg of the Peripheral Vascular Diseases Service of the Norfolk General Hospital, considers that mild lymphoedema of unexplainable origin, in women may be secondary cervicitis. Ascending infection of the pelvic lymph nodes causes central lymphatic obstruction of the leg lymphatics. In such cases clearing

the cervix might afford relief and cure the condition. Vigorous anticoagulant therapy is an essential part of the treatment of practically all types of inflammatory lymphoedema, if the destruction by thrombosis of the lymphatic channels is to be controlled and limited. In its acute stage allergic lymphoedema responds to ACTH and cortisone. In acute iliofemoral thrombophlebitis, the swelling in the legs is usually lymphoedema-

tous. The main lines of treatment in such conditions are anticoagulant therapy and paravertebral blocks of the lumbar sympathetic ganglia. Prolonged rest in bed should never be allowed. Penicillin is of no use. In chronic lymphoedema lumbar sympathectomy is of limited value. Simple paravertebral novocain blocks will effect the same result as regards leg swelling; but lumbar sympathectomy is indicated in causalgia, excessive perspiration, and ischemia.

Only surgical measures will help when chronic inflammatory lymphoedema has caused an indurated leg. Lowenberg performs several types of composite one-stage operations in his clinics. Most of them have as their basis the removal of all pathological skin subcutaneous tissues and fascia; the existing venous pathology is corrected at the same time and resurfacing of the leg with normal skin on normal muscle is also performed. "No matter how horrible a leg appears, there is a normal leg underneath—at the muscle level" says Lowenberg "and with adequate proper surgical management such a leg can be restored to relative normalcy."

Plastic reconstruction of trachea and bronchi.—(*Am. Rev. Tuberc.*, N.Y., 64: pp. 477-479, 1952).

Reconstruction of the bronchial or tracheal lumen may be necessary to preserve lung tissue or life itself, since trauma, tumours, tuberculosis and other infections may obstruct the very lumen. Paulson of the New York Hospital reviews his experimental and clinical experiences with reconstruction of the trachea and bronchi and reports three cases in which he used dermal grafts. The first patient sustained a severe injury in the chest when a heavy drill pipe rolled across his chest and neck. Tension pneumothorax and mediastinal emphysema developed. These necessitated an emergency mediastinotomy and also pleural decompression. The lung expanded well, but seven weeks later, progressive atelectasis became evident. Bronchoscopy and bronchography at this time revealed complete occlusion of the right main bronchus at the carina.

To save the right lung, a plastic reconstruction of the right main bronchus was accomplished by a dermal graft. Narrowing of the bronchus by scar tissue did not interfere with normal pulmonary function.

In the second case in which the patient had tracheal carcinoma, tracheotomy should have been done at the time of the reconstruction, as frequent intratracheal aspirations did not adequately remove the secretions. In the two days before the tracheotomy was performed the lung was irreversibly damaged by the obstruction of the smaller bronchi.

The third patient developed a tracheal stenosis secondary to tracheobronchial tuberculosis. Plastic reconstruction of the upper portion of the trachea by dermal graft was successful but bronchial obstruction was later produced by reactivation of submucosal tuberculosis. The patient died. Bronchoscopy three weeks after the plastic reconstruction of the trachea revealed a satisfactory mucosal lining and the patient had obtained relief from tracheal stridor and exercise tolerance had come back to normal. The author believes that for plastic reconstruction of the trachea and bronchi, wire-enforced dermal grafts are satisfactory.

Indications for and results of splenectomy.—(*Ann. Surg. Philadel.*, 134, p. 815, 1952; *Abst. J.A.M.A.*, 148, p. 673, 1952).

Miller and Hagedorn investigated the records and ascertained the present status of 140 consecutive patients who had undergone splenectomy at the Mayo Clinic from 1942 to 1944; cases in which the spleen was removed as a secondary procedure or for traumatic splenic rupture were not included. Preoperatively 38 of the 140 cases were diagnosed as congenital hemolytic icterus; 47 as essential thrombocytopenic purpura; 45 as Banti's disease or congestive splenomegaly; 5 as acquired hemolytic anaemia; and as "indeterminate splenomegaly." The authors feel splenectomy can be performed for any of the conditions associated with hypersplenism with a reasonably low mortality rate. An exception has a higher mortality

rate than with the blood dyscrasias. In this series of 140 splenectomies there were six deaths, a mortality rate 4.3%. Success of splenectomy for hypersplenic disorders frequently depends on whether accessory spleens have been found and removed. In the event of recurrence of symptoms, especially in thrombocytopaenic purpura, 'the uncertainty of finding splenic tissue at (the second) operation makes a surgical procedure prohibitively dangerous.' In this series, accessory spleens were found and extirpated in 25 cases, or 18%. The two hyper splenic conditions for which splenectomy was most valuable were congenital hamolytic icterus and primary thrombocytopaenic purpura. In about half of the cases of acquired hamolytic anaemia of the primary type, splenectomy produced sustained remissions. Splenectomy is justifiable as a diagnostic measure in the presence of splenomegaly when agnogenic myeloid metaplasia has been excluded by bone marrow studies. Greater emphasis on preoperative bone marrow studies will clarify the indications for, and contra-indications to, operative intervention. The discouraging results of splenectomy for congestive splenomegaly should stimulate further investigation for a procedure of curative value.

Streptomycin in bone and joint tuberculosis.—(Orell, S., *Acta. Chir. Scand. Stockholm*, 102: 113-119, Abst. *J.A.M.A.*, 148: 5, 407).

At the St. George Hospital in Stockholm, 250 patients 31 with spondylitis, 14 with trochanteritis, and 205 with joint tuberculosis, were given surgical treatment along with chemotherapy. The lesions were excised under a protecting "umbrella" of streptomycin and penicillin applied as dry powder (1 to 3 gm. of streptomycin and 200,000 to 600,000 units of penicillin) to the wound. The patients were also given the antibiotics parenterally and PAS orally as per a schedule:—the treatment began and ended with the oral administration of PAS in daily dose of 10 to 14 gm. For 14 days, starting 3 days before the surgical operation, 1 gm. streptomycin divided in 2 doses and penicillin (500,000 units divided in 2 doses or

300,000 units of procaine penicillin in a single dose) were given daily intramuscularly. Administration of PAS was reduced or even completely stopped during the period. The maximum follow-up period has been only 2 years. Primary healing was obtained in all cases. There was no suppuration or sinus formation at all. Tuberculosis did not recur even after surgical intervention in the early stages of the disease.

Radical surgery is effective when streptomycin and penicillin in powder form are applied locally on the wound. The results obtained by Orell suggest that by treating sinuses, bone lesions and joint capsules (synovectomy) it will be possible to shorten the duration of treatment and prevent functional disturbances and relapses.

Late results of thoracoplastic operations.—(*J.A.M.A.*, 148, 5: 395: 1952).

In "Thoracoplasty in the Treatment of Cavernous Tuberculosis of the Lung: A Clinical Statistical Study of Late results," Dr. Erling Refsum reports on the value of thoracoplastic treatment of pulmonary tuberculosis. He compares the patients operated on with those who underwent conservative sanatorium treatment for similar lesions at an earlier pre-thoracoplasty period. The patients undergoing thoracoplasty were operated on in the surgical department of the Riks Hospital in Oslo (Norway), from 1930 to 1939; the observation period was from 6 to 15 years. The controls were patients who had been given conservative sanatorium treatment at the Glittre Sanatorium from 1921 to 1928; their observation period was 18 years or more. Dr. Refsum has succeeded in tracing all the 391 thoracoplasty patients and 105 of the 106 control patients whose lesions and general prospects were, on the whole, more favourable. In spite of this balance in favour of the control group, the death rate among them was 2½ times higher than among the patients undergoing thoracoplasty. Although 70% of the controls died within 12 years 76 per cent of those operated on were able to work

and 50 per cent regained full capacity for work, which in most cases, was heavy manual labour. The results were better for operations on the left than on the right side and better for men than for women. Patients with residual cavities, a year after the operation had a high mortality rate and the results of

bilateral thoracoplasty were disappointing. Of the 25 infants born to women undergoing thoracoplasty 22 were alive and well. Dr. Refsum concludes that women who have become symptom-free after thoracoplasty can undergo pregnancy and confinement without much risk of aggravating the disease.

OBSTETRICS AND GYNÆCOLOGY

The management of obstetrical emergencies.—(*J. Med. Soc. N. J.*, Sept. 1952).

Dr. J. N. Connell of the Margaret Hague Maternity Hospital in Jersey City, describes in a very interesting and informative article, *the modern methods adopted in the management of obstetrical emergencies*.

(1) *Postpartum hæmorrhage*:—This is the most frequent cause of blood loss in obstetrics. It is important too, as a direct cause in maternal mortality and just as a predisposing cause of serious puerperal infections. It is usually due to one of three causes *viz.*, (a) uterine atony; (b) retention of the partially separated placenta or individual cotyledons; and (c) tears of the tissues of the birth tract.

(a) Atony is more frequent in the uterus over-distended by large babies, multiple pregnancies, after prolonged labour, particularly when ill-managed, and after operative deliveries. Ether anaesthesia favours post-partum atony, as also mismanagement of the third stage of labour. At the Margaret Hague Maternity Hospital,—a model institution with all the ideal equipment and abundant expert assistance not matched elsewhere—the bleeding due to atony is controlled by placing the entire hand on the vagina, applying firm pressure against the anterior uterine wall, with the back of the closed hand together with counter-pressure applied by the other hand against the posterior surface of the uterus, abdominally. Ergotrate is injected intravenously and pitocin either I.V. or I.M. The firm uterine compression is maintained until it is evident that the uterus has attained or regained adequate muscle-tone to remain

contracted. The dangerous aspect of postpartum hæmorrhage is that there may be only minimal changes in the pulse and B.P., until large amounts of blood have been lost. Patients can be in a relatively severe degree of shock with normal B.P. and pulse. As soon as atony is evident or is anticipated, a suitably large needle is immediately inserted into a vein if necessary by a quick cut-down, and fluid is given. If the estimated blood loss is 500 c.c. or more a blood transfusion is started immediately. Cross matching should be done at the very onset of the bleeding, if cross matched blood is not ready at hand. Rh-negative processed blood is used and introduced under pressure into the vein. Postpartum loss can often be anticipated. In multiple pregnancy, breech presentation, prolonged labour, all forceps deliveries (except outlet forceps), the patient should be cross matched early in labour and cross-matched blood held ready for use. Intravenous glucose in distilled water should be promptly administered. The bimanual compensation of the uterus, *plus* ergotrate, and blood transfusion will almost certainly result in a return of tone to most atonic uteri. Packing of the uterus is resorted to in some hospitals, early in post-partum hæmorrhage due to atony. But manual compression is definitely better, and two of the three Division Chiefs in the M. H. M. Hospital are vigorously opposed to packing uteri in post-partum hæmorrhage under any circumstances. Dr. Connell however, will pack an occasional uterus when manual compression fails and serious hæmorrhage continues, or until the patient is taken to the operating room for a hysterectomy, which may have occasionally to be done in very

serious cases as a life-saving measure.

(2) *Persistent transverse presentation.*—The management of this condition depends on the state of dilatation and effacement of the cervix, whether or not the membranes have ruptured and the duration of the rupture, the size and shape of the pelvis and the size of the baby. Frequently in the past (and in some clinics even today) where the staff are specially skilled in the performance of internal podalic version, this is the way the problem is solved very often. In the M. H. Maternity Hospital, Jersey City, because of their unpleasant experience with version and extraction, more women with persistent transverse presentation are being delivered by cesarean section. The results are better both for mother and baby. The management of a neglected transverse presentation with an arm prolapsed into the vagina, a shoulder impacted in the pelvis, the lower uterine segment thin, a pathologic retraction ring present and the baby dead, is by decapitation by means of the sharp hook and sickle-knife.

(3) *Prolapse of the umbilical cord:*—This is responsible for a high foetal mortality. In prolapse, the umbilical cord lies below the inlet and lower foetal pole, protruding into the vagina and sometimes even from the vulva. In partial prolapse the cord can be palpated below the pelvic inlet but is above the lowest part of the presenting foetal pole. In cord presentation the cord can be felt through the cervical canal above the inlet.

Cesarean section offers the best chance for foetal survival with a cervix dilated 8 cm. or less and the baby not too compromised. If the cord is weakly pulsating or very slowly, and the foetal heart rate is below 90 or above 160, it is wiser not to hurry into any obstetrical operation which may increase foetal loss. Displacement of the presenting part, Trendelenburg position and anaesthetizing the patient with cyclopropane before attempting hasty delivery will save some babies by controlling anoxia.

(4) *Pre-eclampsia:*—A patient with severe pre-eclampsia is as much an emergency as one who has a prolapsed cord or persistent transverse presenta-

tion. If a severe pre-eclamptic does not show prompt response to treatment, symptomatic and non-specific as it is, termination of the pregnancy is the only thing that will cure it with certainty. Even though many of these babies are premature, they have a better chance of survival in a good premature nursery than *in utero*.

(5) *Rheumatic heart disease in pregnancy:*—A pregnant woman who has certain forms of rheumatic heart disease is often an obstetrical emergency in that she warrants immediate hospitalization and bed rest sometimes for the duration of pregnancy. She requires the care of an obstetrician and a very good cardiologist familiar with this particular problem of rheumatic heart disease complicated by pregnancy.

Nausea of pregnancy—(*Am. J. Obst. Gynaecol.*, Aug. 1952).

A new, effective simple and inexpensive treatment for nausea and/or vomiting of pregnancy is described by Dr. R. L. Merkel of Kansas State. Ascorbic acid and synthetic vitamin K (Menadione bisulphite) were used to decrease placental capillary permeability thereby preventing transfer of the "vomiting factor" to the expectant mother. Merkel used this treatment in a series of 70 consecutive cases, varying from mild to severe. 33 of the 70 women were primigravidas and 37 were multiparæ. All of them received orally 25 mg. ascorbic acid and 5 mg. of synthetic vitamin K every day without any other treatment. Therapy was continued for an average of 30 days until the withdrawal of medication produced no recurrence of the nausea or/and vomiting, 64 patients reported complete remission of symptoms within 72 hours.

Preliminary studies with ascorbic acid alone revealed little improvement in nausea or/and vomiting while the synthetic vitamin K alone showed improvement in about 50 per cent of cases. It was also observed during these studies that oral iron therapy employed in treating anæmia of pregnancy caused little or no gastro-intestinal disturbance and iron absorption was enhanced by vitamin C and K therapy. The prothrombin levels and bleeding time were

determined in every case prior to and after the medication and found to be quite normal.—(*Cur. Med. Digest*, 19:11, p. 109, 1952).

Vaginal length and incidence of dyspareunia following total abdominal hysterectomy—(*Am. J. Obst. Gynaecol.*, 63:400-400, 1952).

In view of the considerable differences of opinion as to the incidence of vaginal shortening and dyspareunia resulting from total abdominal hysterectomy Dr. J. F. Javett of Boston Massachusetts Hospital for Women, studied 61 patients submitted to total abdominal hysterectomy; the vagina was measured preoperatively in all cases; and 51 patients returned for a follow-up study six weeks after operation. A special instrument was used for measuring the length of vagina.

In the 41 patients measured at the follow-up examination, the vagina was either lengthened or showed no change in 36 or 88 per cent, and was shortened in 5, or 12 per cent. A questionnaire in regard to pain on coitus following the operation was sent to 60 women about eighteen months after operation, and 40 replies were received. Of these 40 women, 6 stated they had had no coitus since operation. Of the remaining 34 women, 28 stated that coitus was painless and 5 of them also stated that the operation had relieved their previous dyspareunia. Of the 6 women who stated that coitus was painful 3 said that it was also painful before operation, and 1 that the pain was due to an orthopaedic condition. Thus, the dyspareunia was attributed to the operation by only 2 women (6 per cent).

Therapeutic experiments in female infertility—(*J.A.M.A.*, 148, pp. 603-605, 1952).

The therapeutic value of uterotubal insufflation has been accepted by many writers because pregnancy has occurred many times soon after the procedure. Admittedly the possibility of a psychological influence cannot be dismissed lightly and a true assessment of the method is difficult. Other procedures

involving instrumentation of the cervical canal, such as dilatation of the cervix and hysterosalpingography, have been known for many years to be frequently followed by pregnancy.

A total of 1575 primary sterility patients were followed for 12 months, by Dr. Albert Sharman of Glasgow (Scotland) 893 after insufflation, 253 after the passing of a uterine sound 286 after bimanual examination and 143 after commencing injections of progesterone combined with oestradiol benzoate. The pregnancy rates at the end of three months were fairly uniform (6.9 to 8.4 per cent); at the end of the year the rates were respectively 17.4, 17.8, 10.8, and 19.6 per cent. A much larger comparative study will be required to ascertain the validity of these results.

Dangers of improper vaginal douching—(*Am. J. Obst. Gynaecol.*, July 1952).

Dr. Donald V. Hirst of the J. E. Memorial Hospital, Iowa State has furnished in the *American Journal of Obstetrics and Gynaecology* a broad outline of the techniques and practices that should be adopted in using douches for vaginal irrigation and has also incidentally drawn attention to the dangers in resorting to improper procedures.

(1) Douches should never be given under pressure, if retrograde contamination and bacterial infection should be prevented.

(2) There are two types of douches (a) a warm douche solution for cleansing and hygienic purposes; and (b) a prolonged hot douche using mere hot tap water to apply heat to the pelvic organs.

(3) Douche solutions should always be acid and never alkaline; only then the physiological acidity of the vagina will be maintained or promoted and for this purpose,

(4) the solution should have a pH below 5.0; two teaspoonfuls of lactic acid or $\frac{1}{4}$ cup of white vinegar to half a gallon (2 quarts) of water.

(5) Precoital alkaline douches to promote conception are quite useless and ineffective and are really contraindicated.

(6) Alkaline douches for treating mycotic vaginitis are no more effective than acid douches: the former is also contrary to physiological principles and action.

(7) Acid vaginal jellies are often of greater use than even douches for maintenance of the equilibrium of the vaginal secretion.

(8) Vaginal douches should always be taken, in the lying down posture with the knees drawn up, the hips raised on a heavy folded towel or over the bedpan. The hips should be raised sufficiently high to ensure that the falling away of the intestinal viscera will provide negative pressure to aid in the distention of the vagina by the weight of the solution used for douching.

(9) In taking hot douches, the quantity is not the essential factor but the temperature of the water used. An ordinary half gallon container filled with very hot water, will provide a long enough hot douche of proper temperature and duration, but

(10) the flow should be carefully controlled by the a pinchcock or clamp.

(11) Douches are not effective and cannot be relied upon as a contraceptive measure.

(12) Douches need not be used to ensure hygiene and cleanliness after sexual intercourse provided both parties cleanse themselves with soap and water beforehand. A second cleansing of the parts with clean water after the sex-act.

may be done if the parties so desire.

(13) There is no harm in douching during the menstrual flow, but it is not necessary under normal conditions.

(14) The douche-equipment, container, delivery tubing and nozzle should be cleaned well with soap and water after use and frequently sterilized by boiling.

Libido and hysterectomy.—(Queries and Notes, *J.A.M.A.*, 148:6: '52, p. 500)

A woman whose uterus, ovaries and tubes have been removed continues to have normal satisfaction from and desire for coitus. But her husband assumes she is unable to react normally and therefore, refrains from intercourse. Is the sex urge necessarily terminated by surgery? The answer furnished is:—"The husband is totally wrong in his attitude and since his wife has demonstrated to him the incorrectness of his opinion, one wonders whether this is his only reason for refraining from intercourse with her. Hysterectomy should not and does not lessen libido. As to the role of the ovaries in the human female, the psyche plays a far more important role than the ovaries which play only a very negligible part. Libido is often actually increased after castration or after the menopause. So many auxiliary factors have to be considered, and the husband should discuss the matter with the wife's physician and gynecologist.

BOOK REVIEWS

The Toxemias of Pregnancy—By WILLIAM J. DIECKMANN, S.B., M.D., Mary Campau Ryerson Professor and Chairman of the Department of Obstetrics and Gynecology of the University of Chicago; Chief of Service of the Chicago Lying-in-Hospital and Dispensary; Attending Gynecologist, Albert Merrit Billings Memorial Hospital of the University of Chicago; Associate Editor of the American Journal of Obstetrics and Gynecology. Second edition; with eighty five text illustrations and one colour

plate, 1952, pp. 710. [Published by the C. V. Mosby Company].

This book deals with a disease which is extremely common during pregnancy the detection and proper treatment of which are very important. The author has succeeded in presenting the details of diagnosis and treatment with great accuracy.

The pathologic changes noticed in the disease have been described by the well known pathologist Dr. Sheehan. This enhances the value of the book.

The subject matter is clearly presented and makes easy reading. The book will be found very useful by gynaecologists.

U. V. R.

Oral Anatomy—By HARRY SICHER, M.D., D.Sc., Professor of Anatomy and Histology, Loyola University School of Dentistry, Chicago College of Dental Surgery; Guest Lecturer, Northwestern University, Dental School, Chicago, with 310 text illustration including 24 in colour. Second Edition, 1952, pp. 529. Published by C. V. Mosby Company.

The reviewers of the first edition of this book generally spoke kindly of it. Privileged to read only the second edition, I am convinced that what had been said was no exaggeration. The second edition is said to be a slight improvement on the first, there being an additional chapter on temporomandibular articulation. As the title implies, the book is intended mainly for oral and dental surgeons and students of dentistry and the author has endeavoured to correlate theory with practice.

The first half of this book deals with the basic anatomy of the head and neck while the second half relates to its application in clinical and operational practice—a truly rational approach to the subject. The book contains over 300 beautiful illustrations, 24 of which are in colour. The get-up of the book leaves nothing to be desired, just like all Mosby publications.

U. C. R. B.

Diseases of the Skin—By ROBERT M. B. Mac KENNA, M.A., M.D. (Camb.), F.R.C.P. (Lond.) Physician in charge of the Dermatological Department and Lecturer in Dermatology, St. Bartholomew's Hospital and Medical College, London; Physician to St. John's Hospital for Diseases of the Skin, London; Hon. Consultant in Dermatology to the British Army; Hon. Fellow, The American Medical Association, with a chapter on Radiotherapy by I. G. WILLIAMS, F.R.C.S. (Eng.), D.M.B.E. (Camb.) F.F.R. (Gr. B.) Director of the Department of Radiotherapy, St. Bartholomew's Hospital, London; Consulting Radiotherapist, The Hospital for Sick Children, Great Ormond Street, London. Revision of a

book first compiled by the late ROBERT W. Mac KENNA, M.A. M.D., CH. B. (Edin.) Fifth Edition, 1952, pp. 612, 215 illustrations, 27 coloured plates. Published by Bailliere Tindall and Cox, 7 and 8, Henrietta Street, Covent Garden, W.C.2, London. Price Sh. 42/- nett.

The subject is so vast that it would really be difficult to meet the requirements of a complete textbook on dermatology. The authors have however, produced a very satisfactory piece of work in this Edition. Starting with the detailed anatomy, physiology and pathology of the skin, the methods of examination and diagnoses of various skin conditions together with their treatment are succinctly presented.

This latter portion includes the treatment on general lines and with drugs—both oral and parenteral as also external applications commonly used in skin diseases. The role of diet in treatment, the use of electrotherapy e.g. U. V., rays etc., are also dealt with in this chapter.

Dr. Williams makes a very valuable contribution in the next chapter on the technique and value of Radiotherapy in dermatology. Coccal infections like impetigo and ecthyma, microbial infections such as lupus, leprosy, and anthrax and certain important systemic diseases with skin manifestations like syphilis, leishmaniasis and other infections due to fungi and parasites like scabies, pediculosis etc. are all clearly dealt with in separate chapters. Viral infections of the skin, particularly herpes zoster, which do not ordinarily receive a detailed description in most text-books of medicine, are well presented and have received special attention in this book.

Allergy, drug rash, eczema etc. diseases affecting the hair anomalies of pigmentation, tumours of the skin and diseases of the nails have been given adequate consideration in this book, which is printed in clear bold type on art paper with numerous beautiful illustrative photographs.

This book will doubtless be found very useful not only to the specialists in dermatology but also to the general practitioner.

—U. V. R.

BOOKS RECEIVED

The following books have been received since 15-2-'53 and the courtesy of the Publishers in sending them is acknowledged. Reviews will appear in due course.—Ed.

1. **Transactions of the Fifth American Congress on Obstetrics and Gynaecology**—By GEORGE W. KOSMAK, M.D., 1952. The C. V. Mosby Company, St. Louis. Price \$ 12.50.
2. **Practice of Psychiatry**—By WILLIAM S. SADLER, M.D., F.A.P.A.

The C. V. Mosby Company, St. Louis.
Price \$ 15.00.

3. **The Control of Communicable Diseases**—By HUGH PAUL, M.D., D.P.H., 1952. M/s. Harvey & Blythe Ltd., London. Price 55s. Net.
4. **A Handbook of Radiotherapy for Senior and Post-graduate Students**—By WALTER M. LEVITT M.D., F.R.C.P. (Lond), F.F.R., D.M.R.E. (Camb), 1952. M/s. Harvey & Blythe Ltd., London. Price 30s. net.

CORRESPONDENCE

To The Editor, ANTISEPTIC, Madras.

With reference to the letter of Dr. Kamalapur of Dharwar, published in the December 1952 issue of the ANTISEPTIC, Kaviraj B. K. Shastri, B.M.P., of Tanda, Moradabad writes as under:—"I suggest that the child be given Quinine Sulphate mixture in minute doses, (3 to 5 minims) and then something sour and sweet as lemon drops or orange flavoured sweets. By doing so, the child may be prepared to take foods other than milk only. If this succeeds I would request Dr. Kamalapur to write back and inform the readers through the ANTISEPTIC."

Query

To The Editor, ANTISEPTIC, Madras.

Sir, May I request to be enlightened through the columns of your valued journal on the possible and practicable methods of detecting the offending materials, and treatment (palliative, curative and prophylactic) in a case of allergy e.g., generalised eczematous eruptions with periodic exacerbations and amelioration under antihistaminic or other ordinary treatments?

Harmutty T. E. }
Laluk, P. O. }
Assam, }
14-2-'53. }

D N. DEBSARMA.

NEWS AND NOTES

Higher Studies in T.B.

WHO Fellowships for 4 Indians
(Two S. Indians)

Four Indian workers in different branches of anti-tuberculosis service left by air for Copenhagen (Denmark) on Feb. 14 for advanced study in their specialities under Fellowships, awarded by the World Health Organisation and financed either by UNICEF or U.N. Technical Assistance funds.

Three of the four are at present attached to the New Delhi Tuberculosis Centre. They are Dr. S. P. Pamra, who will study clinical aspects of tuberculosis, Dr. R. Narasimhan, whose fellowship is in the bacteriology of tuber-

culosis, and Mr. Albert Laverne, who will take a course in tuberculosis home visiting.

The fourth member of the group is Dr. T. M. George of Trivandrum, who is at present working at the Tuberculosis Training, and Demonstration Centre, established in Trivandrum in 1951, with assistance from WHO and UNICEF. At Copenhagen. Dr. George will study the epidemiology of tuberculosis, followed by a month's complementary course in London.

Dr. Pamra, and Mr. Laverne will also spend a month in London after completing their six months study in Copenhagen.

Both Dr. Narasimhan, and Dr. George are graduates of the Madras Medical College, while Dr. Pamra did post-graduate work in Madras after graduating in Lahore. Mr. Laverne also received training at the Lahore Medical College. Dr. Narasimhan is the son of Rao Sahib Sri T. N. S. Raghavachari, who was for 29 years Public Health Bacteriologist at the King Institute, Guindy, and who is even now taking keen interest in his old sphere of work as the non-official member of the Government Expert Committee on Water, and Sewage Purification. Mr. Raghavachari has been intimately connected with the ANTISEPTIC and HEALTH since 1947. Dr. Narasimhan on returning in 1945 from active service overseas, served in the King Institute for a few years before he joined the Tuberculosis Centre at Delhi.

During 1952, the World Health Organisation arranged for 76 Indian doctors and health workers to study abroad in a wide variety of medical and nursing subjects under fellowships financed by WHO, UNICEF and U.N. Technical Assistance. Fellowships are granted only to persons specially recommended by the Government and on condition that on return to their home countries, they remain in Government service for at least three years.—“(W.H.O. Release), and *The Mail*.”

Gonorrhoea cases resist treatment with penicillin, streptomycin

New evidence that germs are learning to live with the antibiotics, penicillin and streptomycin, has been found by two French doctors who treated a series of 80 cases of gonorrhoea and reported their findings in the *French Medical Journal, L'Hopital*. However the doctors reported that they did not encounter any cases of the disease which were resistant to terramycin.

Drs. P. J. Viala and T. Groz, state that ten men with gonorrhoea were not cured by doses of penicillin and streptomycin, and that subsequent treatment with terramycin resulted in cures in all the ten cases. Concerning terramycin, they found that in acute gonorrhoea, cases in which terramycin was the first treatment, terramycin-resistance was not found up to the present.

Because of the number of gonorrhoea victims who were not benefited by penicillin or streptomycin, the use of terramycin as the most efficacious in gonococcal infections is recommended.—(M. P. I. B.).

Prolonged treatment; high dosages of terramycin found harmless

The antibiotic terramycin is highly compatible with the body tissues, including the blood, even in very high dosage given over extensive periods of time, recent additions to the literature indicate.

In the most extensive study of its kind to date, 15 tuberculosis patients at Fitzsimmons Army Hospital, Denver, Col., were given seven grams per day of terramycin hydrochloride over a period of 120 days. Twelve additional patients received 2 gm. of streptomycin and 7 gm. of terramycin every third day for 120 days. A close clinical and laboratory watch was kept for the appearance of side reactions, including blood dyscrasias. No evidence of toxicity was found. While some gastro-intestinal irritation appeared in all patients during the first two or three weeks of the treatment, this reaction compelled withdrawal of terramycin in only two cases, despite the massive dosages employed.

Sixty-six tuberculosis patients were given 5 gm. of terramycin daily for 120 days, together with intermittent streptomycin therapy, in a similar study conducted by Dr. Frank L. Miller and his associates. Although approximately 70% of the patients showed some side-reactions, the investigators found that most of them became symptomless except for anorexia after the first sixty days. Only four could not tolerate the full amount of terramycin, and no other untoward reactions were seen. Five grams of the antibiotic daily can be safely administered to virtually all patients.

Recent research reports on the chemical structure of terramycin have shown its molecule to be free from the nitro-phenyl group to which bone-marrow damage from other drugs has been attributed. It is thus anticipated that no such disorders would arise in terramycin therapy.

Numerous additional laboratory and clinical studies have been conducted on the effect of terramycin on the blood picture and it was found that terramycin 'has no influence on blood coagulation at all.'

Confirmation of this conclusion appears in a recent study of the effect of terramycin on bleeding time, clotting time, and prothrombin time in 30 patients, plus observation of "several hundred" additional patients at Harlem Hospital, New York City. On the basis of these tests and observations, Drs. John W. Parker, Jr., and Louis T. Wright, the supervising physicians, declared: "There is no clinical evidence that terramycin produces any change in the blood coagulation mechanism."

Visiting Medical Scientists have moved to Bombay

Smaller Group to visit Delhi

The fourteen-member team of world-famous medical scientists who concluded a brilliantly successful month in Madras (*vide p. 218*) reached Bombay on Thursday March 5th for a strenuous four-week programme with their counterparts at the G. S. Medical College, the Grant Medical College, and the various hospitals and institutes of Bombay.

Five members of the team, including the Chairman, Dr. Samuel Z. Levine, Professor of Paediatrics at the Cornell University, (U.S.A.) may spend four days in Delhi from March 17-22. Two small groups will also go to Poona for periods of three to five days.

In Bombay the Formal Opening of the Team's activities took place in the Convocation Hall of the University on Friday March 6 at 5-30 p.m. with an address of welcome by the Vice-Chancellor, Mr. N. J. Wadia. The opening ceremony was followed by a lecture on "Blood Pressure Regulation" by Dr. Corneille Heymans, Professor of Pharmacology, University of Ghent, Belgium (Nobel Prize winner).

On subsequent days in Bombay, even-

ing lectures, scheduled either at the G. S. Medical College or at the Grant Medical College and open to the whole of the medical and allied professions, will include the following subjects; Advances in our knowledge of Cancer; Septic Wounds; the Epidemiology of Influenza; Aspects of Anaesthesia; Recent Advances in Paediatrics; The Early Diagnosis of Pulmonary Tuberculosis. A number of showings of new medical and surgical films are being arranged. The scientists will spend their mornings with their respective counterparts at the K.E.M. Hospital, the J.J. Hospital, the Medical College Hospital, the Haffkine Institute, etc. Of particular interest are the numerous discussion groups and clinical conferences at which both visiting and resident medical men will take part.

In the team's programme, particular stress is being laid on public health, preventive medicine, and the modern approach to medical education. Lectures and discussions include subjects such as "The Role of the Hospital in the Practice of Preventive Medicine" "The Medical School and the Community" and "Solving a Community Health Problem". There will also be a field study on "Organised Home Treatment for Tuberculosis".

The Administrative Officer of the team has announced that the time of the visitors is fully occupied by their heavy programme in Bombay, and that therefore no requests can be entertained for special visits or consultations. Individual patients cannot be seen except in the course of the team's organised activities in the hospitals and institutions of Bombay, Poona or Delhi.

Dr. Stuart C. Cullen, Professor of Anaesthesiology at the State University of Iowa (U.S.A.), left the team at the end of its programme in Madras, and for the Bombay phase, he has been replaced by Dr. Robert R. Macintosh, Professor of Anaesthetics, University of Oxford (England).—(Based on WHO Press Release dated 4-3-1953).

CORRIGENDUM

"With reference to the advertisement regarding Chinoin Troparin which appeared in page 69 of the February '53 issue of the ANTISEPTIC, the exporters of the above product are Messrs. Medimpex, Budapest VI, Benczur, u. 13 (Bp. 62 P. O. B. 326), Cables: Medimpex, Budapest."

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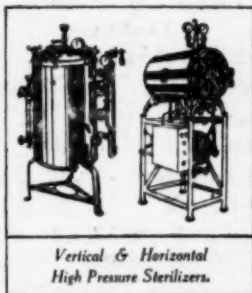
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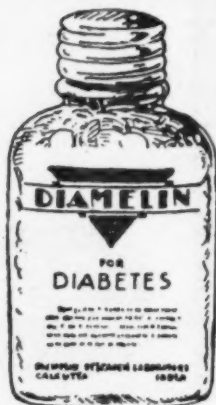
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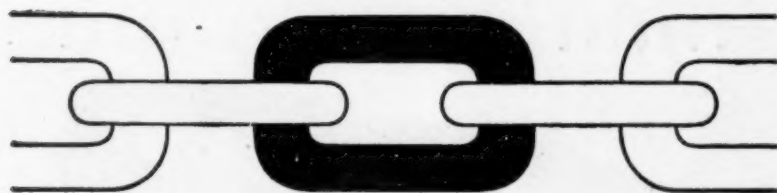
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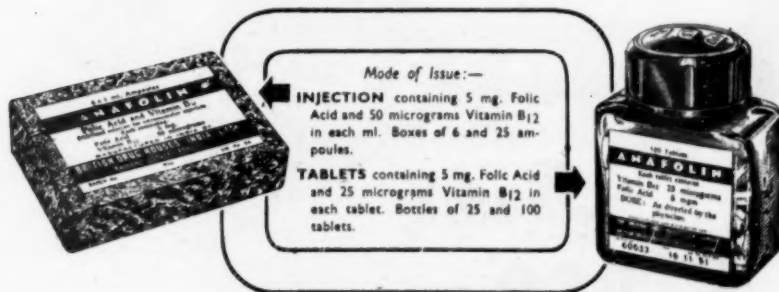
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Cortone—

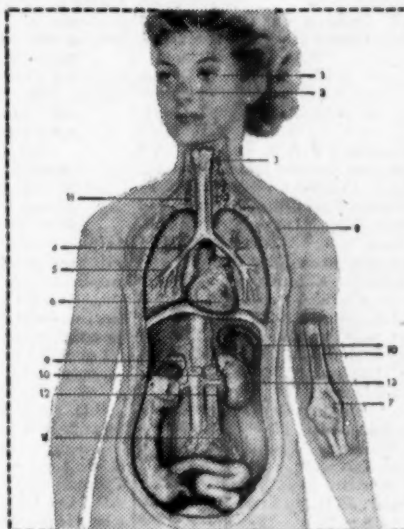
many indications illustrate
its therapeutic importance

CORTONE—original brand of Cortisone—is
available in oral, topical, parenteral forms.

*Primary site
of pathology*

Indications

- | | |
|---|---|
| 1. EYE | Inflammatory eye disease |
| 2. NOSE | Intractable hay fever |
| 3. LARYNX | Laryngeal edema (allergic) |
| 4. BRONCHI | Intractable bronchial asthma |
| 5. LUNG | Sarcoidosis |
| 6. HEART | Acute rheumatic fever with
carditis |
| 7. BONES AND JOINTS | Rheumatoid arthritis
Rheumatoid spondylitis
Acute gouty arthritis
Still's Disease
Psoriatic arthritis |
| 8. SKIN AND
CONNECTIVE TISSUE | Pemphigus
Exfoliative dermatitis
Atopic dermatitis
Disseminated lupus
erythematosus
Scleroderma (early)
Dermatomyositis
Poison Ivy |
| 9. ADRENAL GLAND | Congenital adrenal hyperplasia
Addison's Disease
Adrenalectomy for hypertension,
Cushing's Syndrome, and neoplas-
tic diseases |
| 10. BLOOD
BONE MARROW,
AND SPLEEN | Allergic purpura
Acute leukemia† (lymphocytic or
granulocytic)
Chronic lymphatic leukemia† |
| 11. LYMPH NODES | Lymphosarcoma†
Hodgkin's Disease† |



12. ARTERIES AND
CONNECTIVE TISSUE

Periarteritis nodosa (early)

13. KIDNEY

Nephrotic Syndrome, without
uremia (to induce withdrawal
diuresis)

14. VARIOUS TISSUES

Sarcoidosis
Angioneurotic edema
Drug sensitization
Serum sickness
Waterhouse-Friderichsen Syndrome
† Transient beneficial effects

*CORTONE is the trademark of Merck & Co., Inc. for its brand of cortisone.

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Isonicotinic Acid Hydrazide]	Multivitamin tab. USA 1000 12-8	„ Hicks 3-8; Beacon Flat 1-10
Lepetit 100 3-2; Italy 50 2-4	TCF. Vit. B Complex 6x200 6-0	B.D. Stethoscope 25-8; Ger. 10-0
Dumex Isomex 30's 1-7 100's 3-6	„ „ 1000 5-0; 1x25x100 12-4	Plastic tubing 1-4; Rubber 0-12 yd
Chloromycetin 12 Cap. 20-4 Lqd 14-4	„ W. Liver Ext. 10 cc 3-1	Erkameter 72-0; Aspirin 3-12 lb.
Chloramphenicol Italy 12 cap.	„ „ „ 0 C & B 1000 4-5	Detecto Weighing Machine 42-8
for Typhoid 13-8 Syntemycin 12	„ „ „ Vit. B12 4-12	Saline Apparatus comp 300cc. 8-0
Auromycin 8 Cap. 19-2 [15-0]	„ Folio Acid Comp. 10 cc. 4-12	Wiencarsis large 9-8 [500 cc. 10-8
Terramycin 8 Cap. 16-12; 16 32-0	„ W. Liver Ext. 6x200 3-11	Wall Thermometer Japan 2-0
Combicillin P.S. Pfizer 4-0	Milk Plain 5 cc x100 13-0	Sandoz Cal. Gluco & Vit C
Penicillin c strepto	Milk with iodine amp. 100x500 13-0	5 x 10 cc. 6-8; 10x50 cc. 10-10
Rhodina 2-14 Squibbs 3-4	Alotaris Eng 2-12; Rio 12-8	Sandoz Cal. Gl. 10% 10cc 5amps 5/-
P. D. Camequin Tab. 0-12; Comber	Risochin tab. 10 1-12; 100 14-4	„ „ „ 5 cc. x 10 8-10
Penicillin G Crya. [1000. 6-12]	Redoxen 6x200. 4-12; 50x200. 36-0	„ 50 amp. 44-0 100 cc. x 20 19-0
— 2 5 10 laos.	„ 3x500. 4-4; 25 x 500. 38-0	Estrevisform 20's 2-14; 100's 11-8
— 0-12.6 1-4.6 2-11 Merck	Merck H.T. Emetin 1 gr. orl gr. 3-12	Abu Cotton 1-10; Lint 3-0
— 1-1.0 1-14.0 3-0 Pfizer	Sulpha Tab. 1000 500	Abu Gauze 18 yds x 25" lb. 4-4
„ — 0-11.3 1-3.3 2-1 Govt.	„ nilamide Eng. 11-0 5-10	Bandages 3 1/2 yds. x 1" to 6" 0-9
„ — 1-3.6 2-1 Glaxo	„ guinidine „ 23-0 12-8	Bandages 6 yds. x 3" JJ 4-0 doz.
Strepto c P.A.S. Lepetit 3-8	„ Paok Roy 21-0	Hot water bag 3-4; Ice bag 1-8
Dihydro Strepto Igrm. Glaxo 2-4	„ thiazole Eng. 37-0 19-0	Hypo. Syringe (S.N. Re. 1 more)
Merck, Diamant 1-12; Pfizer 2-4	„ meazathine (100 6-14) 28-8	A.G. Jap. 2 5 10 20 30cc.
Procain Penicillin 20 laos Gl. 4-12	„ diazine USA 75-0; MB 41-8	0-8 0-12 1-0 1-12 2-8
Ind. Govt. Dumex Pf. Gl.	„ Beets 41-8 500; BDH 38-8	Italy 1-2 1-12 2-8 3-8 5-8
4 laos 1-3 1-3.6 2-0 1-3	Sulphathione (100 10-0) 41-4	Germ. 1-0 1-4 1-12 2-12 5-12
3 laos x 10 cc. Oily USA MR	Sulphatriad MB (100 9-0) 44-0	Record Ger. 3-4 4-12 6-8 9-0 12-8
Penicillin Skin Oint 1-8 Eye 1-2	Sulphonamide Bayer 1 lb. 8-0	„ Comp. 6-0 9-0 11-0 —
„ Loxagis 20 1-4 [2 laos x 10 5-8]	„ oream 4 oz. Lilly 5-0 doz	Boston 5-0 5-8 7-0 11-12 15-4
„ Tab. 1/2 laos 12 4-6 1 laos 7-8 PF.	Gentian Violet Jelly 4 oz. Lily 4-4	B.D. Lock 8-4 14-0 15-0 17-0 23-10
„ 1/2 laos Heyden 3-10 1 laos 7-0	Emetine amps. BDH 1 gr. x 12 7-8,	Japan „ 1-9 2-10 3-8 5-0 7-0
PAS Cal. 100 3-5; PAS tab Plain	„ 1 gr. x 12 13-0; 1/2 gr. x 25 13-0	Italy M. case — 2-8 3-4 —
100 3-8; 250 8-4; 500 15-12	„ 6x1/2 gr 3-14; 100x1/2 gr 51-0 box	Ind. „ 1-4 1-12 2-8 3-0
P.A.S. Dumex 100grm 7-4 Ill. 3-10	„ Endo 6 x 1/2 gr. 2-13	Metal case Ind. 50cc. 6-0
„ Quality Ger. 100grm 5-12	„ P.D. 1/2 gr. x 6cc 6-8 1 gr. 9-8	Hypo Syringe 50 cc. S.N.
Quinine Jap. 35-0; Java 51-0	Ksiya Emetin 1/2 gr. x 25 x 100. 10-8	Jap. 4-4; Italy 8-0; Germ. 8-12
„ Holland. 46-0; Howds 55-8	„ 1 gr. x 25 x 100. 18-12	B.D. Luer Lock 29-0; Jap. 9-0
„ oz Jap. 3-0; Howds 4-2	B.W. 1/2 gr. 8-8; 1 gr 7-12	Record Ger. 23-8; Italy 21-0
Q. Bihydro Amps. 100x10grm 2-00.	Cibazol 250 x 13-12; 20's 1-12	Record Needle (Perfectum 5-0)
„ Ind. B.D.H. Evans B.W. P.D.	MB 760 27-12; MB 693 500's 41-4	Jap. Germ. Star. D.B.
„ 15-8 22-8 22-0 32-8 36-8	First Aid Box 11-8 (Amyl Nit. cap 1-8)	1-12 1-10 4-0 4-8 Dz.
„ 10-12 14-8 15-0 100's 5gr. 100.	Vitamin B tab. U.S.A. 500 1-12	All Glass Needles Luer Mount
Euquinine Howd. 4-10; Java 4-10	Liver Ext. 100cc. 2 USP P.D. 3-12	Jap. 2-8 Ger. 3-4 D.B. 5-8 B.D. 10-0
„ Roohe 6-2; Jap. 3-4	„ 5 USP P.D. 7-14	Atehrin Amp 3 grm x 2 2-8 25 16-8
Q. Tab. 2grx 100 2-12; 5gr 4-8 How	Campolan 5x200 5-8; 25x200 25-12	„ 0.1 grm. 6-3-6 [Tooth Forcep 4-8
„ 5 gr. 1400 How. 56-0	Cal. Glu. 10% x 10 cc. 100 13-0	Campbor-in-Oil 3 gr. x 10 cc. x 100
„ Bihydro 2grx 100-12; 5gr 6-12 Rech	Glucose Sol. 25% x 25cc. x 50 10-0	N. Saline 100x500. 6-8 [Cipla 3-12
Pamaquinine 300 Tab. 0-12	„ Thilo Germ. 50 amp. 19-0	Omnonop Amps with Needle
Asparino 1000 Eng. 5-6; Ind. 4-4	Atophanyl I.V. Ger. 5-2; I.M. 5-4	Nivaquin 10 1-12 [Tube 0-8
Mepacrine Eng 1000 11-8; ICI 12-4	Berin 50mg. 3-2; 100 mg. 4-6	F.L. Durex Tin. 2-8 doz. Pkt. 2-0
Quinaorine MB 500 6-0	Calcit Ostalis 1500. 3-3 Entedax 8-0	Ear. Metal Syringe 2oz. 5-0;
„ USA. 5000 52-8; tin 1000 11-8	NAB. 15-0 10-0; 3-0 11-; 45 0-13;	Waterbury Co. 5-2 bot. [4oz. 6-0
Ephedrin 1/2 Gr. 1000 5-4 Germ.	Nicotinic Acid 500 2-4 [6 0-15	Oil Chinapodium oz. 5-0
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Ext. Ergot. 4 oz 4-4 lb 14-8	„ 1000. 6-12; 200. 3-12	Multivitamin tab 1000 E.C. 27-0
Saridon tab. 10 1-8; 250 25-4	Sil. Vit. France 2-12; Protargol	Quinacrin amps 0.3grm tube 0-12
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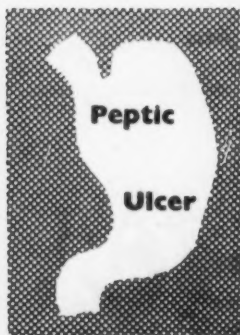
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The negative nitrogen balance in illness leads to breakdown and excretion of body proteins like those of the muscles; the patient gets thinner every day.

HI-NUTRON restores the nitrogen equilibrium preventing waste during illness and aiding convalescence.

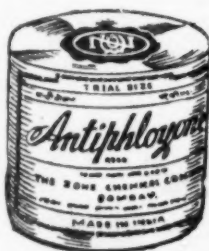
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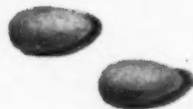
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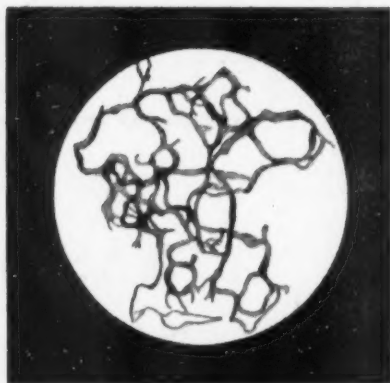
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- Virilisation in the female minimised by the ethinyloestradiol component
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PACKING: Bottles of 30 "Linguets" 2.5 mg. each
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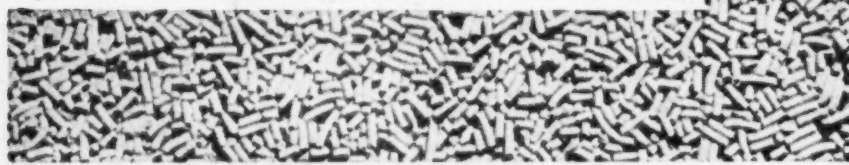
Rapid intestinal absorption and delayed excretion yield exceptionally high, therapeutically efficient P. A. S. levels.

The calcium constituent (about 1.6 gm. calcium daily for 12 gm. P. A. S.) represents a valuable adjuvant.

AMINACYL-GRANULATE WANDER is tasteless, highly concentrated (85% P. A. S. calcium salt) and finely granular, therefore easy to take, and of extremely economical use.

DR. A. WANDER S. A.
BERNE-SWITZERLAND

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" 250 gm. 18-3	" 1-12 1-12 4-0 4-8 ds	" Nivea 7½gr 500 „ 19-2
Auromycetin 8 Cap bot 19-2	All Glass Needles Luer Mount	P.D. Camasuis 3 Tab 0-12-6 pkt.
Acriflavin 25grm 1-12; 5grm 0-9	Jap 2-4; Ger 3-0; DB 5-8; BD 10-8	Penicillin Sodium Cryst. G. :-
Acriflavin 10gm bot 1-0	Hypo Syringe (SN Pe. 1 more)	1 lac 2 lac 5 lac 10 lac
Acriflavin 1000 tabs 3-0	A.G. Jap 2 5 10 20 30cc	0-11-6 0-14 1-6 2-8 Squibb
Atebrin Bayar 300 7-4; 1000 13-8	0-8 0-12 1-0 1-12 2-8	— 0-11 1-3 2-2 Govt.
Adopter Japan doz 2-4	Italy 1-2 1-12 2-8 3-8 5-8	— 0-12 1-4 2-2 Glaxo
Air Cushion I.R. 12 "4-4; 14" 4-12	German 1-0 1-4 1-10 2-8 4-12	Procin Penicillin 20 lacs Gl 5-0
Artery Forceps each 2-4	Recard Ger 3-4 5-0 6-8 9-0 12-8	Ind. Govt. Dumex Squibb Gl.
" " Needle Holder „ 3-4	Boston 5-0 5-8 6-8 11-12 15-4	4 lacs 1-3-0; 1-2-6 1-11 1-4
Aspirin 5gr 1000 tabs 4-4	B.D. Lock 8-4 14-0 15-0 17-0 23-10	Pamaquin 1 gm 500 tabs 1-4
Atophan 20 tabs Tube 2-4	Japan „ 1-8 2-8 3-8 5-0 7-0	Potas Chloras 600 tabs bot 3-12
Berin 1 mg 100 tabs 1-4	M. Cass Ind. 1-4 2-0 2-12 3-4 5-12	PI Mapharside 0.4 gm Tube 1-1
" 500 5 12; 1000 tab. 11-8	Huxley Wintogeno cream 1-10	Pessary Ring 0-6; Check 0-8
Breast Pump 1-8; Bistury 2-0	Injection Eng :-	" Hodges Vul. 0-10
B.P. Blades 6 pkt. 2-8	Camphor Ether o/oil 12xlee 1-4	Quinine Sulph Jap. lb 35-8
" Handle each 3-12	" In oil 12xlee 1-4 box	" Ind. 3-4; How. 1 oz 4-4
BW Atropine Sulph 1/100gr 20 tabs 0-8	Mercury Biniodide 12xlee 0-12	" Bihydro Anpa 100x10grax 2cc
" Digitalin 1/100gr 20 tabs 0-8	" Iodine Rubrum 12xlee 0-12	" Ind. B.D.H. Evans Ger. P.D.
" Hyocine Hydrobrom 1/100	Sodi Glycero Phos. 12xlee 1-4	" 16-8 22-0 23-0 25-8 36-6
or 1/200 gr 20 tabs 0-8	Strychnine Hydro 12xlee 1-4	" 10-12 14-8 15-0 100x5grxl cc
" Emetin Hydro 1½gr 12 tab 5-8	Kit First Aid 12 Units 15-8 box	Q. Tab. 2grxl 100 2-8 5gr 100 4-12
" Quinoxyl 50 tabs bot 1-4	" 24 28-8	" 5gr 1400 How. 55-8
" Emetin Bismuth Iodide 15100tab 13-8	Laxative Vegetable 100 tabs 1-8	" Bihydro 2grxl 100 2-14; 5gr 6-12
Cafin Soda Bezeat 12 amps Eng. 2-8	" 1000 „ 6-8	" HT 5gr 12 tabs tube 1-2
Catheter I.R. 0-8 Ger. 1-4	Liver Ext 100cc 2 USP P.D. 3-13	Quinacrine NE 500 tabs Tis 5-14
" Metal for Male 1-8	" 5 USP P.D. 8-0	Redoxon 100 tabs bot 6-4
" Female 2-4	Litmus Paper Book doz 1-0	Reoschin tab 10 1-12; 100 14-12
Chloromycetin liq. 60 cc 14-0	Leukoplastr 2½x5yds tin 1-12	Rubber Gloves 7½ or 8 1-0 pair
Chloromycetin 12 cap 19-14	3x5 yds 2-8	Roche's Biflavit 250 tab bot 1-4
Cibazol 250's 13-6; 20's 1-10	Merk ½gm Strepto. & Penicillin 2-10	" 1 mg 50x2cc box 2-4
Dental Tweezer 2-4	Pfizer 1 gm 4-0; Squibb's ½gm 3-0	Santonine Synthetic German
Diap. Scale Nick 5-0; Brass 4-0	Glaxo's ½ gm 2-10 Mercks 3-10	1 dr. 3-8; 1 oz 2-4
Dihydro Strepto 1gm Squibb 2-6	M&B 693 1 gm 6 amps box 3-0	Sulphaguanidine Boon 500 tabs 14-8
Merek 1-14; Pfizer 2-4; PD 2-6	" Pracquine 500 tabs bot 1-4	" thiazole „ 500 „ 20-8
Glaxo 2-0; A.H. 1-14	M&B Neptal 6xlee box 2-6	" Diazine „ 500 „ 41-8
Dumex Iaconex 25's 1-5; 100's 3-5	Meonine Wyeth USA 100 tabs 24-8	Sulphatriad 25 2-2; 100 tab 8-8
Elasto Plaster 2½ x 5 yds tin 3-0	Murine Eye Remedy 2-12	Sulphamezathine 3cc 25amp 7-8
" 3 x 5 yds „ 4-0	NAB 15's 0-10; 3 0-11; 45 0-13;	Stethoscope pouche Plastic 1-8
Emetin Hydro ½gr 6xlee PD 7-8	Nicotinic Acid 500 2-4 [6.0-15	" with Handle 2-0
" „ Endo ½gr 6xlee 3-0	Neosalvarsol 0-15 30 45 60grm	" with two Zips 6-8
" „ BDH ½gr 12xlee 12-8	(60gm 1-6) 0-14 0-15 1-1 1-3	Suture Needle Eng. 0-4 each
" „ BW ½gr 12xlee 8-12	Oil Chinopodium 1 oz 5-2	Squibb Hydrazide 25's 1-15; 100 4-14
" „ ½gr 6xlee 8-4	Ointment :-	Thermometer Germ 0-15; Jap 0-11
Enterovisform 20's 2-12; 100's 11-10	Acid Boric USA 1 oz 0-4 each	" Zeal 2-10; USA 1-6; Eng 1-5
Ephedrin Hydro Ger ½gr. 1000 tabs 5-6	" „ 4 oz 0-8 „	" Hicks 3-8; Jap Flat 1-8
Eye Bath Glass each 0-10	" Salicylic 1 oz 0-5 „	TCF. Vit. B Complex 10cc 5-0
Finger Stall doz 1-0	Acriflavin Boon's 2 oz 0-5 „	" W. Liver Ext. 10cc 3-2
F.L. Washable 0-5 each	Atropine ½ oz 0-12 „	" „ o C & B 10cc 4-6
" „ Crocodile 0-8	Blue USA 1 oz 0-5 „	" „ o Vit. B; 10cc 4-12
" „ Silvertex 2-4 doz	Calomel USA 1 oz 0-8 „	USA Bandages Brown 3x6yds 0-6
F.L. Durex Pkt. 1-12 Tin 2-0 doz	Cibazol Eye Oint 1-4 „	" „ 2x1½ yds 0-3
Folic Acid 5mg 25 tabs USA 48-0	" Skin 2-6 „	" „ white 3x6 yds 0-6
Glass Pan 0-10; Rods 0-3	Gentian Violet Jelly 4 oz 0-7 „	" „ First Aid 4 x 1½ „ 0-4
" „ Syringe 1 oz 0-8; 2 oz 0-10	Mercurial „ ½ oz 0-5 „	" „ Triangular 24x48 Ft. 1-0
" „ 4 oz 0-12	Melphanilamide 4 oz 0-8 „	Vitamin B; USA 500 tab 1-8
Glucose Sol. 25% x 250cc 50amp 15-0	PAS German 100 gm 5-8	Voramon 10 1-6; 20 tabs 2-7
Glykeron Smith USA 3 oz 9-4	" Calcium Ger. 100 gm 5-8	Vitamin C 1000 tabs USA 18-0
Hypo Syringe 50cc S.W.	" „ Ger 7½gr 100 tabs 3-8	Weight Set Dr. & Gr. 0-12
Jap. 4-8; Italy 8-8; Germ. 8-8	" „ 7½gr 250 tab 7-8	Yeast 5gr 1000 tabs 5-4
B.D. Luer Lock 31-8; Jap 9-8	" „ Holland S.C. 7½gr 100 tab 3-8	" 7½gr „ 7-0
Record Ger. 23-8; Italy 21-0	" „ „ 250 „ 8-0	Zambuk Ointment 1-2
Record Needle (Perfection 5-8)	" „ „ 500 „ 15-4	

JAPAN PARKER TYPE FOUNTAIN PEN 4-12 Each.

Transfusion Simplified



CROOKES (COMPLETE UNIT) TRANSFUSION

SIMPLICITY

The unit comprises the 20 oz. (560 ml.) transfusion bottle and the sterilized Accessories Set — nothing else. The bottle is fitted with a specially designed bung and occlusive metal cap; the accessories include every item needed for operating the unit.

SPEED OF ASSEMBLY

Assembling consists of nine simple actions which can be performed in a few minutes once the sequence of events has been understood. The importance of this vital time-saving needs no stressing.

NEW TYPE BOTTLE

It will be noted that the new type bottle is a considerable improvement on the old swing stopper type which is now discontinued.



THE CROOKES LABORATORIES LTD

(Incorporated in England)

COURT HOUSE CARNAC RD. BOMBAY 2



Control of Hypertension

Successfully achieved with

RALFEN

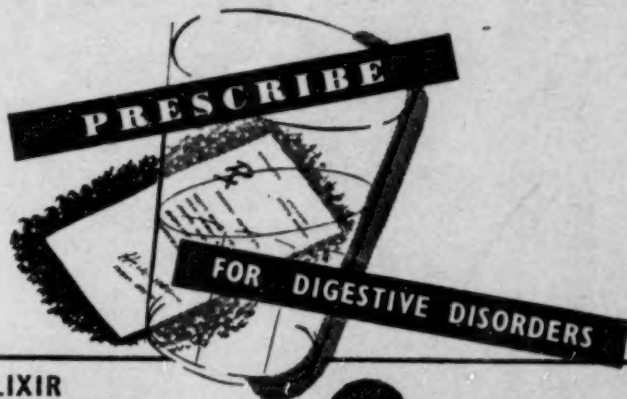
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STANDARDIZED EXTRACT OF RAUWOLFIA SERPENTINA

- REDUCES HIGH BLOOD-PRESSURE
- ACTS AS A SEDATIVE TO NERVOUS SYSTEM
- USEFUL IN INSOMNIA, EPILEPSY ETC.
- INDICATED IN MENTAL DISORDERS OF MANIACAL TYPE.

Supplies : Liquid Extract : In one ounce phials.
Tablets of 5 grs. In bottles of fifty.

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CALCUTTA : BOMBAY : KANPUR.



ELIXIR DIGENZYMES

Palatable combination of
Diatase, Pepsin and Papain



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IN THE
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CHLOROMYCETIN has one of the widest antibacterial spectra among established antibiotics and, in addition, is available in several forms. Its remarkable activity against a great number of pathogenic organisms—bacteria, rickettsiae and viruses, gives it a wide application in the field of tropical medicine.

Chloromycetin has been used successfully in the treatment of

AMOEBIASIS · BOUTONNEUSE FEVER · DYSENTERY · TRACHOMA · TROPICAL ULCER · TULAREMIA
 TYPHOID AND PARATYPHOID · TYPHUS AND SCRUB TYPHUS · UNDULANT FEVER
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*Supplied in vials of 12 kapseals of 250 mg.**

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PÆDIATRIC Chloromycetin PALMITATE

A pleasant-tasting suspension of a bitterless derivative of the antibiotic for administration to children. One teaspoonful (4 c.c.) is equivalent to 125 mgm. Chloromycetin. **Bottles of 60 c.c.**

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A cream indicated in the treatment of pyodermas, folliculitis and dermatoses of infective origin. Also effective as a routine minor wound dressing. **Tubes of 1 oz.**

FOR OPHTHALMIC USE

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A buffered, stable ophthalmic solution indicated in the treatment of bacterial and viral conjunctivitis, trachoma, keratitis and herpes zoster ophthalmicus. **In vials of 15 c.c. capacity.**

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A petrolatum-base oculentum of 1% Chloromycetin, for the topical treatment of conjunctivitis and other eye infections.

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